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Table of Contents

ORIGINAL ARTICLES— Page

An Address: All of One Company, by G. T. Gibson 321
Weight Gain in Pregnancy, by L. O. S. Poidevin .. 324
Thalassæmia Major, by J. G. Wilson 328
"Self-Sterilization" of Chemically Treated Blankets, by Sydney D. Rubbo, Bryan C. Stratford and Shirley Dixson 330
Agoraphobia: A Review of Ten Cases, by B. C. Bignold 332
A Comparison of the Diets of Two Groups of School-Children, by Audrey Cahn and Rosemary Hepburn 333
Anti-M Antibody as a Rare Cause of Hæmolytic Disease of the New-Born, by R. T. Simmons, Vera I. Krieger and Rachel Jakobowicz 336
Warm Blood for Exchange Transfusion in Infants, by W. H. Kitchen 338

REPORTS OF CASES—

Myocardial Infarction Complicated by Severe Hæmorrhage from a Spontaneous Laceration of the Stomach (Mallory-Weiss Syndrome), by M. A. Mishkel and R. Jeremy 340

REVIEWS—

Hypnosis in Anesthesiology 343
Peripheral Facial Palsy 344
Pediatric Pathology 344

BOOKS RECEIVED 344

LEADING ARTICLES—

The Genetic Basis of Thalassæmia 345

CURRENT COMMENT—

The Transmission of Leprosy 346
Doctor Jenner of Berkeley 347

ABSTRACTS FROM MEDICAL LITERATURE— Page

Ophthalmology 348
Oto-Rhino-Laryngology 349

BRITISH MEDICAL ASSOCIATION—

South Australian Branch: Annual Meeting 350
Annual Meeting of the British Medical Association, Auckland, February, 1961 355

OUT OF THE PAST 356

CORRESPONDENCE—

It Happened One Night 356
Specialists and Hospitals 357
Organization of the Profession 357
The Detection of Advanced Cancer 358

NAVAL, MILITARY AND AIR FORCE—

Honours 358
Appointments 358

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA 359

NOTES AND NEWS 359

NOTICE—

New South Wales Association for Mental Health and National Guidance Council of Australia .. 360

NOMINATIONS AND ELECTIONS 360

DEATHS 360

DIARY FOR THE MONTH 360

MEDICAL APPOINTMENTS: IMPORTANT NOTICE .. 360

EDITORIAL NOTICES 360

An Address.[1]

ALL OF ONE COMPANY.

By G. T. Gibson,

President of the South Australian Branch of the British Medical Association.

This is the annual occasion when a medical audience hears a doctor speak on a subject which is not of clinical interest, and it is probably the only occasion when I shall so speak. Accordingly it is appropriate that I should try to express the thoughts which twenty-five years of practice and membership of this Association have brought. I have used the words of Sir Francis Drake as the title of my address in order to stress the basic importance of intra-professional unity.

A previous president, mentioning the quaint ordeal of the presidential address, advised me to pick a good text and preach a brief sermon, and you will be reassured to learn that I hope to ignore this advice and avoid the obvious pitfalls, though when I finish my remarks you will probably feel that several texts would apply.

[1] Read at the annual meeting of the South Australian Branch of the British Medical Association on June 29, 1960.

This evening I want to speak to you of the very great changes in medical practice that have happened in my own professional lifetime and are still happening, and in this I do not intend to refer to the great changes and advances in scientific and clinical medicine, but to the whole social background of modern medical practice.

Medicine as an organized profession—or, if you prefer it, doctors *en bloc*—has never been a popular institution. I believe that individual doctors are more often than not liked and frequently respected by their own patients; but this is a very different thing, and does not alter the basic fact that in general the public is at least suspicious of medicine as an entity, and this has probably always been the case. Imhotep was deified 4000 years ago; but if one had access to current comment, I am sure there would have been criticism to be heard around Karnak.

Closer to home, Chaucer gives us a clear picture, in his portrait of his "Doctor of Physic", of the popular opinion of the doctors of his day; but it is in the highly disputatious seventeenth century that one sees a protracted, if entertaining, picture of medicine at bay, for it was in this century that medicine began to emerge from the hibernation of the Middle Ages and become a real factor in ordinary living. Picture the doctor of those days; he had no real knowledge, and there was no basic science for him to build on. Such training as there was at the universities was in the classics and dialectics. He had to be a formidable debater, and for the rest his whole

career was based on sheer bluff. Sydenham's injunction to go to the bedside was a startling innovation in that age.

The century was one of most undignified quarrelling between the doctors, as exemplified by the Royal College of Physicians, the surgeons and the barber surgeons and the apothecaries. Into this arena toss a large selection of quacks, and then add brisk arguments with Court, Parliament and public opinion, and one can imagine a highly disputatious if futile era of medicine. Pamphleteers abounded in all camps, and produced a harvest of sheer vituperation which will probably never be equalled. Not all were attacks of one faction on another; many were personal attacks of doctor on doctor where theorists disagreed. This *odium medicum* provided much ammunition for the detractors, and it is to be doubted whether the influence of this era is even yet fully lost.

Then as now, the public were prepared to respect solemnity only if it was backed by real knowledge and skill, not if it was based on pretence. The seventeenth century doctor was repeatedly assailed for his pretentions, his pedantries, his quarrelsomeness and his covetousness, and it is interesting to see that much criticism of our own profession in these days is based on these same old points of debate.

Late in the seventeenth century, the Royal College of Physicians urged its members to give free advice to the poor. Even if this act was originally a tactical move in the war against the apothecaries, it was another step towards the establishment of the modern trend in medicine with its free hospitals and honorary system, and it was the first admission of a moral obligation and an ethical responsibility to see that none should die or suffer needlessly when medical knowledge could save or relieve.

I have related these historical facts mainly to show that the real problem of the doctor and his social background has always existed, and that we face no new problem. The pattern is continuously changing, and our responsibility is to ensure the place of doctors in the community—or, if you prefer it, to ensure that the public relations of the profession are given full consideration and are sensibly and skilfully handled.

As an over-simplification, it is fair enough to state that our predecessors of earlier centuries were vulnerable because they were ignorant in an age of ignorance, whereas more recently our closer predecessors were more happily placed, in that they had a degree of knowledge soundly based in a community which was quite uneducated in such matters, and therefore more prone to pay respect.

Our own problem is different again; we are highly trained and highly skilled in a group of sciences whose range has increased enormously, but we live in an age where general enlightement is much greater and where our function is therefore again more vulnerable to criticism.

Let us, in the light of these modern conditions, review our positions in terms of the points of debate mentioned a few minutes ago. I think we can profitably consider pretensions and pedantries under the same heading. Probably there are very few, other than the inevitable cranks, who would seriously doubt the knowledge and skill of the modern doctor. Most responsible people acknowledge and respect this; on the other hand, they also recognize that there are degrees of mental acuity and skill among doctors. Most of our patients nowadays have had some elementary instruction in physiology, at least sufficient to read and understand the articles on medical topics which appear in popular papers and digests; accordingly, in our dealings with patients we must be prepared to respect their intelligence as they respect ours. There are no ivory towers of mysticism to which we can retreat, and I for one regard this as a highly desirable change.

One criticism levelled against us is that we do not explain to the patient either the nature of his illness or the rationale of its management. Very few patients expect to understand the complications and niceties of their illness, but they do feel they can understand a straightforward and simplified explanation, and surely they are entitled to it. It is so easy when one is overworked and tired to brush aside the patient's questions; but by doing so sensible cooperation may be lost. The hurried exposition deliberately given in technicalities to confuse the issue is an insult.

Some of my colleagues have told me that, although ideal in theory, it is actually a waste of time to explain disease to patients, and quote the highly garbled accounts of previous trouble one gets on taking a history. This is often so; but I wonder how much of what has been said to them has been perfunctory and over-involved. I am sure that most surgeons will agree with me that it is often of great value if the patient knows what a pre-existent scar really represents.

I am convinced that the relics of medical mysticism must disappear from our relations with patients, and that an open sensible relationship will benefit both. We are no longer a fraternity whose calling sets us apart from humanity, but a highly specialized scientific profession living and working in an age of scientific enlightenment.

There is another difficulty in the doctor and patient relationship, and this is the over-scientific approach—if you will pardon a phrase, the wonder-drug attack. I am not decrying modern therapy, but the tendency to rely excessively on potent antibiotics or potent ataractics can work to the detriment of accurate diagnosis. It can be bad medicine, and it can ignore the patient's real needs. Basically, this tendency depends on consideration of the disease or even the symptoms only, whereas in actual fact there is always a patient attached to the disease. My age group and my seniors will readily remember the old-fashioned penumonia with its old-fashioned crisis. There was, in actual fact, in those days no treatment for the disease; but it is surprising in retrospect how much could be done for the patient. Here I would plead with teaching staff to stress and re-stress the need to afford ease and comfort to the patient while the disease is dealt with, and the means whereby this can be done.

A third trend of modern medical practice which is having a profound effect on the doctor-patient relationship is the development of group practices. Here, again, we can see a factor that can make the doctor impersonal, and present medicine to the public in the guise of a technique unrelated to the personalities involved. The advantages of group practice to both doctors and patients are obvious and need no recapitulation; however, certain safeguards are necessary, and extra vigilance is needed by the doctors. On many a public rostrum in times of public debate we have stressed the patient's right to free choice of doctors, and it is essential that this choice must not be limited by the organization of a clinic, and that the patient can ask to see whatever member of the clinic is preferred, and to see another member at a later date if he so desires. From complaints and criticisms freely given in the public Press, it would appear that this freedom is sometimes withheld in group practices. It devolves on the partners in a group to see that the patient is not arbitrarily channelled, though after the patient has selected the doctor there should be very little subsequent difficulty. One of the dangers of group practice is that it is very easy for too much administrative power to pass into the hands of a secretary or receptionist without the medical partners ever realizing the fact. Public comment has been made on this also.

It should be relatively easy to show the public that group practice can provide efficient medicine and a continuous cover of safety in emergency. It is the responsibility of those involved in this type of practice to prove to their patients that the proper personal relationship can be maintained, and that the girl who manipulates the telephone and appointment book has no dictatorial powers.

Let us revert to the quarrelsomeness with which our predecessors were accused. No longer do we issue pamphlets, bills and broadside to scarify our colleagues and amuse the public; but are we in fact as reasonable and united as we should be? Are the mutual criticisms

between our internal groups always strictly factual and unbiased?

The mere fact that the range of medical knowledge is so wide and is still widening brings its own problems. The golden age of the doctor-patient relationship was in the recently-past day of the family doctor, when it was possible for one man to carry out all reasonable procedures and so in himself satisfy the medical need of his patients single-handed. Now the wide range of specialization shows beyond cavil that there are many divisions of our science, demanding additional training and acquired skills which can absorb a man in a lifetime of unremitting work. This is realized, not only by doctors, but also by the public, which has accepted specialization as an essential part of its medical protection, and more and more is tending to demand the appropriate specialist in times of major problems—a fact which some general practitioners resent and some try to ignore. Yet, if the specialist is important to the patient at the rare and critical times, the general practitioner is essential at all times, and is not relegated to an insignificant or minor role.

Disunity within the profession or antagonism between branches of it can only work to the detriment of us all. Frank and critical discussion is necessary and desirable for the interchange and development of ideas; but discussion if it becomes sectional or personal reverts to uselessness or is actively damaging. There is only one standard to adopt, and that is the greatest benefit to the patient. This alone will suffice to ensure that debate never degenerates to quarrelling, and a sincere recognition of this one fact must eliminate all friction from the relations between the various branches of medicine.

We have always been, and probably always will be, accused of covetousness. This lies near the core of practically every attack made on us today. In the public mind there seem to be three aspects of this: firstly, statistical tables which show that doctors are in the highest income group in the Commonwealth; secondly, the impact of medical fees in time of serious or prolonged illness; thirdly, the amount of honorary service given by doctors. What is the real position? This question is closely bound to the future of medical practice; we must be quite clear in our own minds where we stand.

If it is a fact that we are highly paid, how far is this justified? Commonly it is accepted that there should be a relationship between reward, effort and responsibility. The facts are that the doctor begins to earn only after a minimum of eight years of minimal reward. His initial expenses and sacrifices are greater than the average. He is dependent solely on his own efforts, and must provide for his own old age without the benefits of pension or retiring allowance. He accepts throughout his working life great limitations on his personal life, and shoulders great responsibilities. He works hours far in excess of the ordinary. In general his expectation of life is reduced; therefore a higher income seems quite justifiable, provided that this income is paralleled by the service given by the doctor. In this respect we have to consider, not only the skill and efficiency involved, but also a reasonable accessibility on the part of the doctor, who has to perform or provide for the performance of his duties at all times and under all circumstances. This is another very prevalent criticism of us at present, and one of which we must take notice. It is no answer to the critics to say that the doctor is entitled to recreation, as this was never the point at issue.

Regarding the impact of individual fees on the patient, one would feel that the patient who has had the foresight to take out insurance in accordance with the current scheme of medical benefits would be protected from undue hardship. This is probably the case, and our responsibility is to ensure that fees are commensurate with the services performed and with the income of those receiving them. Much that is said in this respect comes from people who have been too improvident to insure themselves or whose political philosophy supports socialization of medicine. In spite of political creeds or of national schemes of medical insurance, it is our duty as well as our interest to maintain the traditional altruism of the medical profession.

In my opening remarks I referred to the changing face of medicine, and I have endeavoured to show the essential change necessary in our public relationships to bring medicine into its rightful place in the social background of modern life. It is my belief that this is desirable from the viewpoint of patient and doctor, and that in its truest sense it is ethically right. However, if there should be disagreement, let me point out that it is indicated also baldly and bluntly by pure self-interest.

Throughout the world the march of events is towards socialization and State control. There must come the time when a government will wish to enforce State control of medicine in Australia, and when that day comes we shall have to accept it, reject it and fight it or agree to accept a modified scheme. If the day comes for the profession to fight a government, the profession will need to be able to mobilize a large force of public opinion behind it and also to be firmly united itself. The questions which will mould public opinion on socialized medicine are those of costs and efficiency of service. Only the most ingenuous could regard State medicine as "free", in view of the published rising and astronomic costs of similar schemes which have had to be met by taxation. However, the public will not be worried at the thought of doctors being salaried as civil servants or controlled by bureaucrats, so long as they feel that they will get at least as good a service as that provided now. Now is the time to consolidate good public relations and show our patients that the present relationship is human and efficient beyond what could be expected from bureaucratic medicine. At the present juncture the practitioner who is indifferent, non-cooperative, unavailable or aloof is doing the patient, his colleagues and himself the greatest disservice of which he is capable. It is against this background that we are engaged in the formation of the Medical Association of Australia—an autonomous body designed to be fully representative of our profession in this country, whose function it is to correlate and control our policies and to maintain our true and legitimate standards. It is not seen either as a trade union or as a political weapon, but as an organization dedicated to the maintenance of professional efficiency and integrity. Many of us who have enjoyed membership of the British Medical Association for so many years regret the severance while accepting its inevitability and desirability, and we are determined to maintain full affiliation with the Parent Body. That we are taking this step with the blessing of the British Medical Association is a mark of the maturity of Australian medicine.

The new Association must be the product of thought, responsibility and dedication, as it is destined to develop and extend its influence over generations and centuries far into the future. As with the old Association, the qualifications for membership will be simple. Anyone who is a legally qualified and registered medical practitioner in good repute, so that he can find a proposer and seconder, can join.

Within our own ranks we have many Colleges, societies and associations representative of groups of doctors practising in this or that speciality. Many of us are proud to be members of such Colleges, and very rightly so, and we gladly pay our tribute of loyalty to them. Nevertheless, the more limited loyalty cannot override the wider one, since however rarified may be the specialist atmosphere of any doctor, he is still basically a medical man dedicated to the care and relief of humanity, and as such he is one of a great army and not merely a member in a *corps d'élite*. We must make very sure that this is the basis on which we form our new Association—that it will be the centre of corporate medical life, representative fully of general practitioner and specialist, of young and old, of the obscure and the prominent, never influenced by sectional interests either geographically or from within our own subdivisions, fully representative of the doctor.

To this end I would urge particularly our younger members to take a direct interest in the affairs of the

Association as an integral part of their career, and not merely as an umpire or a refuge in times of stress. It is a rewarding interest. I have found, as many of my predecessors have, that the man who accosts one to complain of blundering or mismanagement or worse on the part of the Council is usually a member who takes little interest in our problems and seldom is seen at a meeting. One of the constant difficulties your Council has to face is that of promulgating information and obtaining expressions of general opinion. The solution of this is in your hands, not ours.

In the new Association I would most strongly urge you to see that selection of your representatives is wisely made, so that all colours of medical opinion gain free expression and that administration of your interests lies in capable hands. This is the basis of strength of such an Association.

There is only one body which can represent the medical profession in Australia, and that is the Association which soon will be the Medical Association of Australia. When it comes to matters of national importance, only the great representative association can speak, and it is vitally necessary for the welfare of the profession and the public that the new Association should have, not only the full support of the individual members, but also that of the Colleges and societies. The Medical Association of Australia must inherit from the Parent Body the tradition of authority; it must be the custodian of medical ethics and morals; and it must be the voice with which the whole profession speaks on matters of unusual medical importance and on medical policy.

At the beginning of this address I declined to give you a text. Now I should like to point my remarks by giving you two. The first is from the Koran, the seventeenth Sura, "The Night Journey":

> For his own good only shall the guided yield to guidance, and to his own loss only shall the erring err.

The second is from the Bible, the Book of Ecclesiastes:

> Whatsoever thy hand findeth to do, do it with thy might.

WEIGHT GAIN IN PREGNANCY.

By L. O. S. POIDEVIN, M.D., M.R.C.O.G.,
Reader, Department of Obstetrics and Gynæcology, University of Adelaide.

THE present-day tendency for a rather over-enthusiastic interpretation of weight gains in pregnancy suggested a study of more than eleven hundred women reporting to the Queen Elizabeth Hospital (Maternity).

Electrolyte variations in so-called normal and toxæmic pregnancies continue to present controversial and often unexplained findings. For example, a recent study (Dieckmann and Pottinger, 1956) showed that in normal pregnant women the sodium content of both the rectus muscle and the abdominal wall skin was increased, yet in those with pre-eclamptic and hypertensive pregnancies it was decreased. Again a clinical review of 2077 cases by Robinson (1958) demonstrated not only the prophylactic value of extra salt, but also a remarkable improvement in toxæmic women when they were fed 200 to 300 grains of salt daily. Many explanations have failed adequately to explain these and other anomalies which in themselves expose the need for a better understanding of this intricate problem. This present study does not concern itself with electrolytes.

Causes of Weight Increase in Pregnancy.

Weight gain can be considered under three headings:

1. That due to the products of conception, together with the normal maternal physiological changes such as increased blood volume, and uterine and breast hypertrophy.
2. That due to water retention.
3. That due to fat deposition.

An increase of 24 to 26 lb. is generally accepted as about the upper limit of gain in normal cases. However, there are many examples of normal pregnancies in which the total weight gain has been far greater or far less than this average. Excessive weight due to water retention should be lost rapidly after delivery, which should thus help to differentiate it from adiposity.

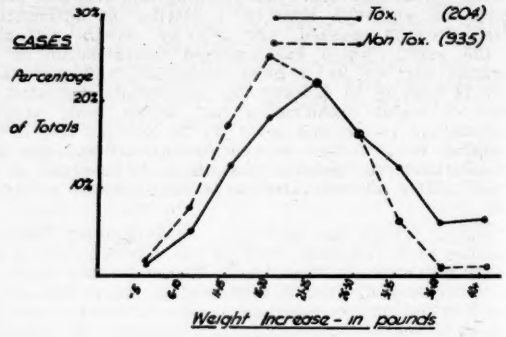

FIGURE I.
Absolute weight gains—whole of pregnancy.

Evidence is available to support pregnancy, *per se*, as a direct cause of obesity (Sheldon, 1949). In a small series of more than one hundred primigravidæ recently studied in detail, it was found that 70% remained heavier than their pre-pregnant weight six months after delivery (Poidevin, 1959*a*). Obesity is an integral sign of Cushing's syndrome, and there is much laboratory evidence in support of a Cushing-like stimulus of pregnancy (Venning, 1946; Gemzell, 1953; Robinson *et alii*, 1955; Jailer, 1956; Gold, 1957; McKay *et alii*, 1957) as well as clinical evidence (Browne, 1958; Poidevin, 1959*a* and 1959*b*).

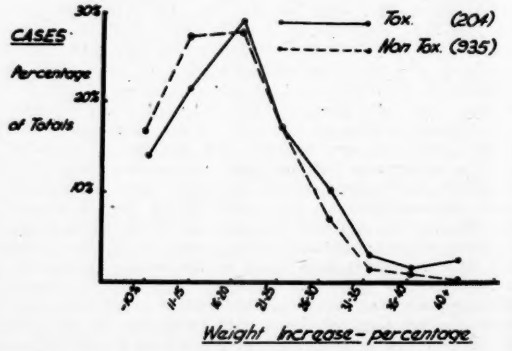

FIGURE II.
Percentage weight gains—whole of pregnancy.

Previous Studies.

One of the objectives of the present study was to evaluate weight gain in relation to normal and toxæmic pregnancies. Whilst it was in progress, Fish *et alii* (1959) reported their findings from a study of 1000 pregnant women. Of their normal group 72% gained excessive weight, in accordance with ordinary standards, and they concluded that excessive weight gain and toxæmia should be largely dissociated. Chesley (1944) stated that 72% of their toxæmic patients had weight gains of less than 30 lb. Siddall and Mack (1938) reviewed 100 cases of toxæmia and concluded that ". . . the occurrence of

excessive weight gains in pregnancy would appear to be of doubtful significance in predicting impending toxæmia and of secondary value, at most, in the diagnosis of the actual disease", and further, ". . . so far as toxæmia is concerned, we do feel that the significance of excessive weight gain has apparently been misunderstood or greatly overrated". Woodhill *et alii* (1955) noted a relationship between pre-pregnancy obesity and toxæmia, although no association was found between excessive weight gain during pregnancy and the incidence of toxæmia. Hamlin (1952) stressed the importance of a rate gain in excess of 8 lb. between the twentieth and thirtieth weeks of pregnancy as prognostic of the later development of toxæmia.

More recently (1960) the same author has stated that the diagnosis of mild toxæmia may be made on the presence of excessive weight gain and œdema. No doubt the application of this criterion explains the very close relationship which he showed between absolute weight increase and toxæmia. In primiparæ his toxæmia rate rose from 8% to 50% in parallel with weight increase. In multiparæ his rate rose from 3·8% to 21% over the same range of weight increase.

If weight increase is to be closely linked with the diagnosis of toxæmia, then it will be impossible to study one independently of the other. It appears to me that in these studies, the diagnosis of toxæmia should be made

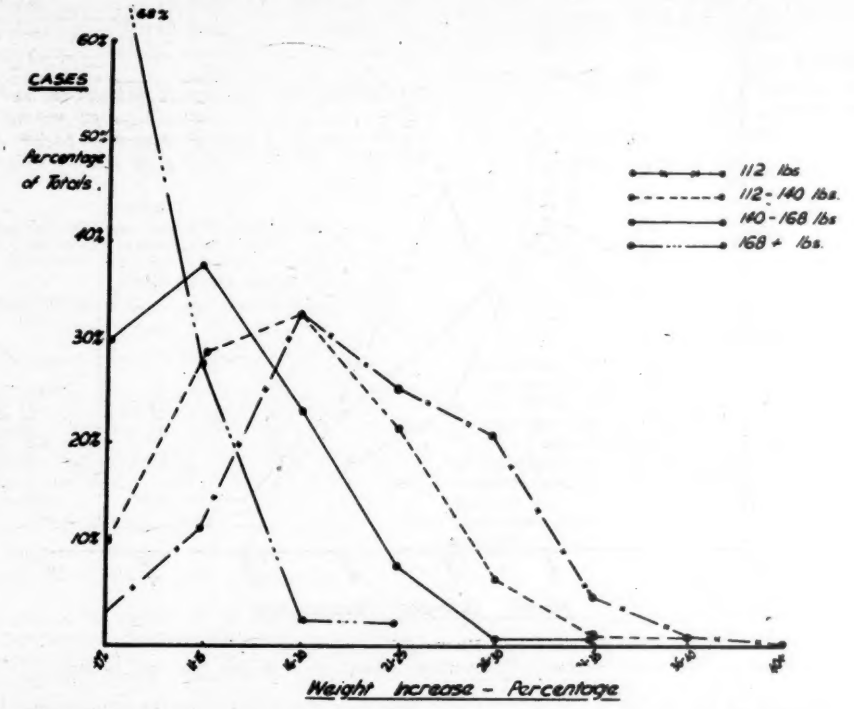

FIGURE III.

Weight at the first visit related to percentage weight gain in 935 non-toxæmic subjects.

F. J. Browne (1946), after reviewing the work of many authors, concluded: "On the whole, therefore, provided too much is not expected from it, a case appears to have been made out for routine weight taking in pregnancy". Cummings (1934) reported that 131 women from 1000 studied gained more than 30 lb. throughout pregnancy; 63 of these developed toxæmia — that is, 48%. Thus the majority of those gaining excessively did not develop toxæmia.

The Diagnosis of Toxæmia.

The diagnosis in borderline cases is obviously one of the weaknesses in studying this question. Those studies in which the investigators have enthusiastically diagnosed toxæmia show a rate which would not be generally acceptable, with the result that an unreal relationship appears between weight gain and toxæmia. Stevenson (1952) made a detailed study of the out-patient records of "500 consecutive normal patients" of the Women's Hospital, Crown Street, Sydney. These 500 women consisted of 144 who weighed over 9 st. 7 lb. early in their pregnancies, and 356 who were under 9 st. 7 lb. One hundred and fifteen of the heavier women and 172 of the lighter women developed toxæmia—that is, 287 out of 500 consecutive women, an incidence of 57·4%.

without undue emphasis on weight gain. The cases in this present study were selected in accordance with the following criteria.

Toxæmias.

1. Hospital admission was a basic criterion for toxæmias. Women showing excessive weight gains and treated entirely from the ante-natal clinic were not accepted in the toxæmia group.

2. Severity was graded as mild, moderate or severe. (*a*) Mild cases were those in patients showing some degree of relative hypertension and œdema or excess weight gain, who were admitted to hospital for observation, responded well to therapy and were permitted to go to term. (*b*) Moderate cases were those in patients causing more concern because of reluctance to improve with strict therapy, whose previously normal blood pressure did not rise above 160/90 mm. of mercury; these patients may or may not have shown albuminuria. (*c*) Severe cases were those in patients with either persistent hypertension above 160/90 mm. of mercury or persistent albuminuria, whose condition necessitated induction of labour.

Two hundred and four cases fitting the above criteria were selected consecutively from clinic patients of the

Queen Elizabeth Hospital who booked in before the twentieth week of pregnancy. In order to collect this number in this manner, the records from October, 1954 (opening of hospital), to July, 1958, were closely examined.

Non-Toxæmias.

Nine hundred and thirty-five cases in patients who booked before the twentieth week of pregnancy, and who did not develop toxæmia, were selected from the Queen Elizabeth Hospital records. They were selected consecutively from serial numbers 1 to 1775, beginning from October, 1954. Ante-natal and in-patient management remained standardized over this period of time. Thus a total of 1139 cases was studied.

Table I shows the period of greatest weight gain in both the normal and the toxæmic subjects. (Only in 829 of the full sample of normal subjects was this information recorded.)

A greater percentage of the non-toxæmic subjects showed a more rapid rate of weight gain in the 20-30 week period than did the toxæmic subjects. The toxæmic subjects showed the more rapid gain from the thirtieth week onwards.

D. Weight at First Visit Related to Percentage Gain.

The weight at first visit related to percentage gain was analysed for both the non-toxæmic and the toxæmic subjects (Figures III and IV). The lighter the woman at the first visit, the greater was the relative gain in weight. The heaviest women showed the smallest percentage gains. These findings applied to both the toxæmic subjects and the controls.

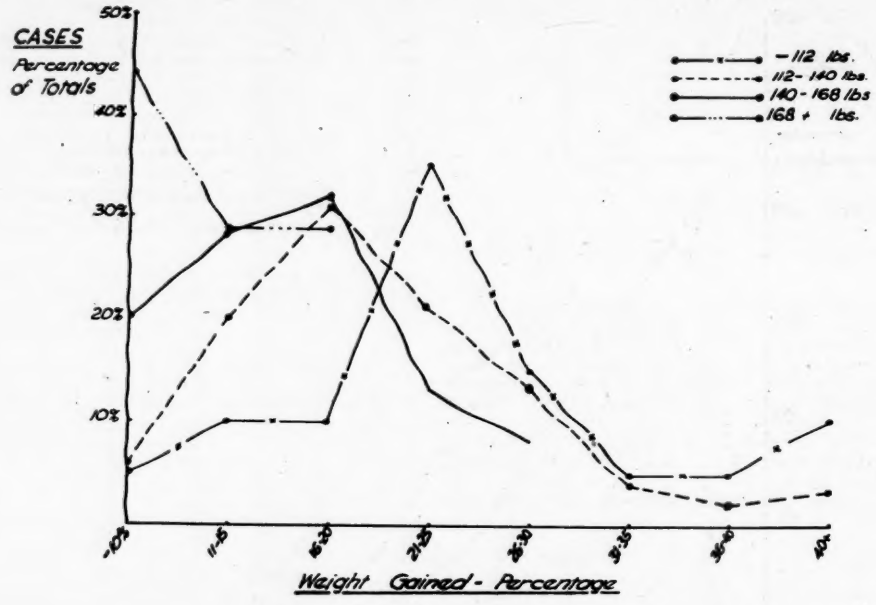

FIGURE IV.
Weight at first visit related to percentage weight gain in 204 toxæmic subjects.

Results of the Study.

A. Absolute Weight Gain: Whole of Pregnancy.

The absolute weight gains in both normal and toxæmic pregnant subjects are shown in Figure I. The actual number of cases in each group has been converted to a percentage in order to obtain comparative curves. In the higher increment groups the toxæmic subjects prevail, in the lower increment groups the normal subjects are more prevalent. However, in the 21-30 lb. increase range there was an equal prevalence, namely, 40%. In 24% of the toxæmic subjects and 10% of the normal subjects the gain was in excess of 30 pounds (Chesley, 1944, showed 28% of toxæmic patients gained in excess of 30 pounds).

B. Percentage Weight Gain: Whole of Pregnancy.

When the absolute weight gain was converted into a percentage of the initial weight, the curves shown in Figure II were obtained. Note that the pattern is similar to that in Figure I, so that either way of reporting weight gains seems to yield much the same result.

C. Period of Greatest Weight Gain.

In each woman the rate of weight gain was analysed in relation to two periods of pregnancy, namely, that from the twentieth to the thirtieth week, and that from the thirtieth to the fortieth week.

E. Weight at First Visit Related to Absolute Gain.

Table II shows only the figures for the highest and the lowest initial weight group (228 subjects — toxæmic patients and controls).

The lighter the initial weight, the greater is the absolute gain. The heavier women show a smaller absolute gain. Thus again the similarity of expressing weight gains in either relative or absolute amounts is demonstrated.

F. Height Related to Weight Gain.

There was a slight tendency demonstrated for taller women to gain less absolute and relative weight than those in the smallest group.

G. Weight Gain Related to Length of Labour.

Table III contains information recorded in 1121 cases. Toxæmic and non-toxæmic subjects are shown separately.

Table III shows a doubtful correlation between the shorter labours and the smaller percentage weight gains. Alexander and Downs (1953) suggested such a relationship from their study, although they did not consider it statistically significant.

H. Weight Gain Related to Severity of Toxæmia.

In Table IV the peak of the absolute weight gain is seen to be approximately the same in each of the three groups. Thus no specific relationship could be demonstrated between the weight gained and the severity of the toxæmia.

TABLE I.
Period of Greatest Rate of Weight Gain.

Period of Greatest Gain.	Subjects.	
	Non-Toxæmic (829).	Toxæmic (204).
20th to 30th week	614 (74%)	129 (63%)
30th to 40th week ..	215 (26%)	75 (37%)

Eclamptics.—There were seven booked eclamptics, whose total weight gains ranged from 5 lb. to 36½ lb. Only two

TABLE II.
Weight at First Visit Related to Absolute Gain (228 Subjects, Toxæmic and Control).

Weight Increase (lb.).	Weight at First Visit (lb.).	
	168+	112−
5 (or less) ..	4	0
6 to 10 ..	15	6
11 to 15 ..	17	21
16 to 20 ..	12	50
21 to 25 ..	8	43
26 to 30 ..	3	23
31 to 35 ..	2	10
36 to 40 ..	1	2
40 and over	0	1
Total ..	62	166

of the seven gained in excess of 20 lb. throughout the whole of the pregnancy. From seven cases no conclusion can be permitted; however, there was no tendency for those patients who developed eclampsia to gain excessive

TABLE III.
Percentage Weight Gain Related to Time in Labour.

Weight Gain (Percentage).	Time in Labour (Hours).					
	Less than 12.		12 to 24.		24 and Over.	
	Toxæmic Subjects.	Non-Toxæmic Subjects.	Toxæmic Subjects.	Non-Toxæmic Subjects.	Toxæmic Subjects.	Non-Toxæmic Subjects.
10 (or less) ..	20	113	8	38	2	7
11 to 15 ..	25	187	3	52	5	14
16 to 20 ..	36	187	17	67	5	9
21 to 25 ..	19	100	9	47	5	19
26 to 30 ..	11	47	5	17	5	2
31 to 35 ..	3	10	1	2	1	1
36 to 40 ..	1	2	1	2	0	1
40 and over ..	2	0	2	0	0	1
Total ..	117	646	56	225	23	54

weight. It has been shown previously that eclampsia is related more closely to cerebral dysrhythmia than to the degree of toxæmia (Rosenbaum and Maltby, 1943; Macintosh, 1952; Poidevin, 1955).

I. Toxæmia Related to Blood Group.

An incidental and quite unrelated observation concerning the relationship between toxæmia and blood group was observed during this study (Table V). This investigation was done because of recent interest shown in this relationship. The frequencies recorded are approximately the same as those found in the general population. The early work of Pike and Dickins (1954) suggested a higher frequency of toxæmia among group O women. Their later work (Dickins *et alii*, 1956) denied this.

No specific relationship between the incidence of toxæmia and any particular blood group results from this study.

TABLE IV.
Weight Gain Related to Severity of Toxæmia.

Weight Increase (lb.).	Grade of Toxæmia.		
	Mild.	Moderate.	Severe.
5 (or less)	0	1	1
6 to 10	3	5	1
11 to 15	7	9	9
16 to 20	19	11	7
21 to 25	21	12	13
26 to 30	16	10	9
31 to 35	10	9	7
36 to 40	7	4	1
40 and over ..	3	6	3

Discussion.

This study of weight gain in pregnancy shows a higher percentage of toxæmic women in the upper increment group. This finding is neither new nor unexpected. Of the members of the toxæmic group, 24% gained more than 30 lb., while 10% of the non-toxæmic controls did this. From Figure I it is clear that 40% of women in both the toxæmic and the non-toxæmic groups gained total weight within the range of 21 to 30 lb. Within this range, therefore, there is absolute similarity between the two groups.

TABLE V.
Toxæmia Related to Blood Groups.

Blood Group.	Toxæmic Subjects.		Non-Toxæmic Subjects.	
	Total.	%	Total.	%
O	85	41·7	441	47·0
AB	7	3·4	23	2·5
B	23	11·3	91	9·9
A	89	43·6	380	40·6

The rate of weight gain during two 10-week periods of pregnancy failed to show any prognostic importance between a more rapid weight gain in the 20- to 30-week period and subsequent toxæmia. In fact, increased rate of gain in this period showed a slight comparative statistical immunity to toxæmia. No prognostic importance either way can be deduced from these findings.

Group studies such as this, and many others already mentioned, indicate the unreliability of considering excessive weight gain as diagnostic of toxæmia or even subsequent toxæmia.

Excessive weight gains in pregnancy are less likely to be due to the products of conception than to either water retention or fat. We have yet to learn to differentiate between fat deposition and water retention short of œdema. The stimulus to fat deposition during pregnancy is well known, and there is no evidence to deny that each individual is entitled to her own variations in the total amount and the rate of fat deposition. A study recently conducted at the Queen Victoria Maternity Hospital has

shown a greater reluctance on the part of toxæmic women to return towards their pre-pregnant weight at six weeks *post partum* than those in the normal group. This study, which involved only 246 women, showed that 52% of toxæmic women and 66% of non-toxæmic women returned to within 5 lb. of their initial weight. This suggests that adiposity is at least playing a part in both groups, but perhaps even more so in the toxæmic women.

A study at present being conducted at the Queen Victoria Maternity Hospital, under standardized conditions, is demonstrating how common it is to find a daily variation in weight from 2 to 3 lb. in pregnant women in hospital wards for conditions other than toxæmia. Thus until such time as we have a finer appreciation of the real cause of these variations in weight, there would appear to be little justification for any confident prognostic import as concerns the individual.

In this study the severity of the toxæmia has been shown to have no relationship with the amount of weight gained. In the eclamptic group itself only two of the seven had weight gains in excess of 20 lb.

There appears to be no advantage in the conversion of absolute to relative weight gains, for in the sections of this study which considered both the results appeared almost in parallel.

The incidental blood group relationships were studied because the opportunity was presented. No specific relationship with toxæmia was shown.

Conclusions.

1. Weight gain in pregnancy may equally well be considered absolutely or relatively.

2. The toxæmic subjects as a group showed a tendency to greater weight gains than the non-toxæmic controls, except in the range from 21 to 30 lb., when 40% of both groups gained equally. In 24% of the toxæmic subjects and 10% of the non-toxæmic subjects the gain was in excess of 30 lb.

3. Increased rate of weight gain between the twentieth and thirtieth weeks of pregnancy showed no prognostic value in relation to subsequent toxæmia.

4. The lighter the initial weight, the greater was the relative and absolute weight gain. This applied to both the toxæmic subjects and the controls.

5. The taller women gained less relative and absolute weight.

6. There was no real evidence to show that smaller percentage weight gains were associated with shorter labours.

7. No specific relationship was demonstrated between the severity of toxæmia and the weight gained.

8. No specific relationship was demonstrated between the incidence of toxæmia and any particular blood group.

9. Weight gains in pregnancy are still not understood in sufficient detail to justify more than the broadest conclusions in each individual. At present there are no reliable clinical means of distinguishing between episodes of fat deposition and water retention. Such episodes of excessive gains indicate the need for the closest observation.

Acknowledgements.

I wish to record my gratitude to Miss Jennifer Burden, of this department, for the punch-card recording of all data, and my thanks to the other members of the honorary staff for the review of patients under their care.

References.

ALEXANDER, S. A., and DOWNS, J. T. (1953), "Influence of Weight Gain on Pregnancy", *Amer. J. Obstet. Gynec.*, 66: 1161.

BROWNE, F. J. (1946), "Antenatal and Post-Natal Care", 6th Edition, Churchill, London: 369.

BROWNE, F. J. (1958), "The Ætiology of Pre-eclamptic Toxæmia and Eclampsia", *Lancet*, 1: 115.

CHESLEY, L. C. (1944), "Weight Changes and Water Balance in Normal and Toxic Pregnancy", *Amer J. Obstet. Gynec.*, 48: 565.

CUMMINGS, H. H. (1934), "An Interpretation of Weight Changes during Pregnancy", *Amer. J. Obstet. Gynec.*, 27: 808.

DICKINS, A. M., RICHARDSON, J. R. E., PIKE, L. A., and ROBERTS, J. A. F. (1956), "Further Observations on ABO Blood Group Frequencies and Toxæmias of Pregnancy", *Brit. Med. J.*, 1: 776.

DIECKMAN, W. J., AND POTTINGER, R. E. (1956), "Etiology of Pre-eclampsia—Eclampsia", *Amer. J. Obstet. Gynec.*, 71: 596.

FISH, J. S., BARTHOLOMEW, R. A., COLVIN, E. D., GRIMES, W. H., LESTER, W. M., and GALLOWAY, W. H. (1959), "The Relationship of Pregnancy Weight Gain to Toxemia", *Amer. J. Obstet. Gynec.*, 78: 743.

GEMZELL, C. A. (1953), "Blood Levels of 17 Hydroxycorticosteroids in Normal Pregnancy", *J. clin. Endocr.*, 13: 898.

GOLD, J. J. (1957), "Blood Corticoids: Their Measurement and Significance: A Review", *J. clin. Endocr.*, 17: 296.

HAMLIN, R. H. J. (1952), "The Prevention of Eclampsia and Pre-eclampsia", *Lancet*, 1: 64.

JAILER, J. W. (1956), "Adrenal Function in Pregnancy", *Bull. Sloane Hosp. Wom. N.Y.*, 3: 82.

MACINTOSH, R. R. (1952), "The Significance of Fits in Eclampsia", *J. Obstet. Gynæc. Brit. Emp.*, 59: 197.

MCKAY, E., ASSALI, N. S., and HENLEY, M. (1957), "Blood Levels of 17-Hydroxycorticosteroids during Labour of Human Pregnancy", *Proc. Soc. exp. Biol (N.Y.)*, 95: 653.

PIKE, L. A., and DICKINS, A. M. (1954), "Blood Groups and Toxæmia of Pregnancy", Brit. *Med. J.*, 2: 321.

POIDEVIN, L. O. S. (1955), "An Electroencephalographic Study of Patients with Toxæmia of Pregnancy and Controls", *J. Obstet. Gynæc. Brit. Emp.*, 62: 417.

POIDEVIN, L. O. S. (1959*a*), "Normal Pregnancy: A Cushing's Syndrome", MED. J. AUST., 2: 149.

POIDEVIN, L. O. S. (1959*b*), "Striæ Gravidarum: Their Relation to Adrenal Cortical Hyperfunction", *Lancet*, 2: 436.

ROBINSON, H. J., BERNARD, W. G., GRUBIN, H., WANNER, H., SEWEKOW, G. W., and SILBER, R. H. (1955), "17, 21-Dihydroxy-20 Keto-steroids in Plasma During and After Pregnancy", *J. clin. Endocr.*, 15: 317.

ROBINSON, M. (1958), "Salt in Pregnancy", *Lancet*, 1: 178.

ROSENBAUM, M., and MALTBY, G. L. (1943), "Cerebral Dysrhythmia in Relation to Eclampsia", *Arch. Neurol. Psychiat. (Chicago)*, 49: 204.

SHELDON, J. H. (1949), "Maternal Obesity", *Lancet*, 2: 869.

SIDDALL, R. S., and MACK, H. C. (1938), "Weight Changes and Toxæmia of Late Pregnancy", *Amer. J. Obstet. Gynec.*, 36: 380.

STEVENSON, R. B. C. (1952), "Some Aspects of Pre-eclampsia and Eclampsia", MED. J. AUST., 1: 317.

STEVENSON, R. B. C. (1960), "Toxæmia of Pregnancy", MED. J. AUST., 1: 282.

VENNING, E. H. (1946), "Adrenal Function in Pregnancy", *Endocrinology*, 39: 203.

WOODHILL, J. M., VAN DEN BERG, A. S., BURKE, B. S., and STARE, F. J. (1955), "Nutrition Studies of Pregnant Australian Women", *Amer. J. Obstet. Gynec.*, 70: 987.

THALASSÆMIA MAJOR.[1]

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THE purpose of this paper is to report a case of thalassæmia major and to review briefly our experience of the condition at the Adelaide Children's Hospital.

With the increasing numbers of people of Mediterranean origin migrating to Australia, the specific trait associated with thalassæmia is being seen more commonly. The trait is being found with increasing frequency particularly among the patients attending the main maternity hospital, where the Greek and Italian mothers comprise a large percentage of the patients. Under the stress of pregnancy these persons become anæmic. The anæmia is typically recognized as a hypochromic anæmia which does not respond to iron therapy. The anæmia is of all grades of severity, and some patients need blood transfusion as part of their ante-natal treatment.

[1] Read at a meeting of the South Australian Branch of the British Medical Association on April 31, 1960.

The disease of thalassæmia occurs in two forms, the major and the minor form, although there are probably all gradations between the two (Smith, 1943). The major form was first described by Cooley (1925, 1927, 1932), and is a severe, chronic, progressive anæmia occurring in childhood, characterized by five main clinical features, which will be described later. Early reports in Australian literature include those of Sinn (1949), who described the case of a boy, aged nine years, and of Baker and O'Neill (1951), who reviewed the condition up to that time.

Since 1955, seven patients with severe thalassæmia have been seen here and their families investigated. The accompanying tables show very briefly some of the features of these families. Only six cases have been labelled thalassæmia major. All these patients have attended hospital before the age of one year, looking desperately pale. The child in Case III we did not see until she was aged four and a half years, but she had had treatment elsewhere from an early age. It is to be noted that three of the six cases have presented in the first three months of this year, so it would appear that the incidence of the disease may be increasing quite rapidly. The Adelaide Children's Hospital is the only children's hospital in South Australia, hence our figures should be representative of the incidence in this State.

Another point to be noted is the high fœtal hæmoglobin percentage in all cases. This estimation, therefore, from our point of view, can be used as a criterion for diagnosis in these severely affected infants.

The case presented below is the most recently seen case of thalassæmia major, and illustrates most of the classical features as seen in this disease.

Report of a Case.

The patient was a male child aged 11·5 months, whose parents were of Greek extraction and lived in the city of Adelaide. The mother noticed that the child had been pale for about three months. He had been examined by his local doctor several times for minor complaints and given an iron mixture and several tonics. Twenty-four hours prior to his admission to hospital, the child had been feverish and irritable and had vomited several times. He was the only child. He had never had any immunizing injections. The birth and neonatal period had been uneventful. His birth weight was 6 lb. 14 oz. and his weight at the time of examination was 22 lb. He had developed quite normally with regard to age milestones. The parents had been in Australia for only two years. The father came from Sparta and had always been well. The mother came from Kalme and had always looked a little pale. During this pregnancy she became severely anæmic and required two blood transfusions. She had had no other pregnancies or miscarriages.

Examination of the patient showed him to be a very pale Greek infant, with a temperature of 100° F. and a pulse rate of 180 per minute. He was in good nutritional state. There was no lymphadenopathy. The liver was palpable one finger's breadth below the right costal margin, and the spleen was grossly enlarged to three or four fingers' breadth below the left costal margin. There were no other abnormalities. A blood examination gave the following information: hæmoglobin value, 5·8 grammes per 100 ml.; erythrocytes 2,950,000 per cubic millimetre (reticulocytes 5%); leucocytes, 17,900 per cubic millimetre, polymorphs 34%, lymphocytes 57%, monocytes 2%, eosinophils 2%, neutrophil metamyelocytes 2%, neutrophil myelocytes 3%). There were 13 nucleated red cells in counting 100 white cells. The result of the Coombs test was negative. The child's blood was Group A, Rh-positive. Fœtal hæmoglobin made up 31% of the total hæmoglobin. The blood film showed great variation in size of the red cells from microcytes to macrocytes. They also showed marked pallor. Poikilocytosis and target-cell formation were noted.

The diagnosis of thalassæmia major was then made. A sample of blood from both parents was examined, and it was found that they both carried the thalassæmia trait. Neither was anæmic at the moment, although the mother became severely so during her pregnancy.

After some discussion it was finally decided to give the child a blood transfusion. He was, therefore, given 400 ml. of blood by scalp-vein transfusion, and the hæmoglobin value on his discharge from hospital was 12·7 grammes per 100 ml.

Discussion.

The diagnosis of this condition usually becomes obvious by the time the child is about nine months of age. Patients are usually pale prior to this, but it is not usually noticed. They are naturally liable to infections, and it is commonly because of one or repeated infections that these children present first to the doctor, when the extreme pallor may be noticed incidentally. A good account of the clinical features is given by Baker and O'Neill (1951), who stress the following five main clinical features of the condition: racial incidence, familial incidence, splenomegaly, hæmatological changes and skeletal changes.

The majority of cases described have been in people of Mediterranean origin, as in all of our cases. However, apparently authentic cases have been reported in other races. Both parents of severely affected children show features of the minor form of the disease as is shown by our cases, the details of which are as follows:

Family I:

Father	Minor
Mother	Minor
Tina	Minor
John	Minor
Tony	Normal
Jimmy	Major

Family II:

Father	Minor
Mother	Minor
Susan	Minor
Jonay	Normal
Hassan	Major

Family III:

Father	Unknown
Mother	Unknown
Cutos	Minor
Nick	Minor
Irene	Normal

Family IV:

Father	Unknown
Mother	Unknown
Aleki	Major

Family V:

Father	Minor
Mother	Minor
Guiseppe	Normal
Michele	Major

Family VI:

Father	Minor
Mother	Minor
Maria	Normal
Anita	Major

Family VII:

Father	Minor
Mother	Minor
Nicholas	Major

It is thought that the minor disease represents heterozygote carriers of the trait, and that the severely affected children represent homozygous forms of genetic inheritance. However, the genetics of this condition have not been completely worked out as yet, and this is a fascinating field for further study.

Splenomegaly was a constant feature in all our cases. The blood pictures in all our cases were essentially similar to that in the case described. In assessing the significance of a high fœtal hæmoglobin percentage in this age group, it is important to remember that the normal adult range (0 to 1·7%) does not pertain. At birth the fœtal hæmoglobin represents about 30% of the total hæmoglobin present, and this level slowly falls, so that by the age of nine months there should be very little left. The hæmoglobin value in these children is an interesting feature. It appears to find a basal level often between 4 and 6 grammes per 100 ml., where it remains. It would seem that at this level blood formation can keep pace with blood destruction in these children. After a blood

transfusion it is commonly observed that the hæmoglobin level quickly falls to this basal level again, where it remains until it may fall even lower or infection overtakes the child and further transfusion is necessary. The decision for transfusion in these children is a difficult one. Infection will overtake the child if it is left for long with a hæmoglobin value of 5 grammes per 100 ml.; on the other hand, the effect of repeated transfusions in these children is invariably the development of an acquired hæmolytic anæmia as a result of blood-matching difficulties. It is from this complication that most of these children apparently die. The patient in Case I, after 17 transfusions, is entering this phase now, and the question of splenectomy is under review at the moment.

TABLE I.
Cases of Thalassæmia Major.

Case Number.	Age when Patient was First Seen at Adelaide Children's Hospital.	Date.	Fœtal Hæmoglobin.	Transfusions.
I ..	8 months.	October, 1956.	37%	17
II. ..	6 months.	November, 1958.	78%	4
III ..	4 years 8 months.	May, 1959.	61%	4
IV ..	4 months.	February, 1960.	82%	2
V ..	1 year.	March, 1960.	88%	1
VI ..	1 year.	March, 1960.	31%	1

The skeletal changes are an interesting feature of this condition. These are the result of continuous marrow hyperplasia from early life, and are seen sometimes in the facial features of the child and also radiologically. Early changes are seen particularly in the metacarpals, where a widening of the medullary cavity is associated with trabeculation. In the skull there is widening of the diploë followed by radial striation in the parietal and frontal regions, the so-called "hair standing on end" appearance. These skeletal defects, in association with the pale, muddy-yellow colour of some of these children, contribute to the mongoloid facies often described (Caffey, 1937, 1951). In Case III in our series this facial expression was present.

In reviewing our cases, the most important point in regard to practical management is exemplified in Case I. Here we have now performed 17 blood transfusions. Naturally we have added considerably to the iron content of the body; but also, in this case, we are running very short of available veins for transfusion purposes. In this regard it was comforting to read of the apparent success of intraperitoneal transfusion as a method of treatment in these cases (Newman, 1959).

Summary.

The purpose of this report has been to bring this condition to the notice of practitioners, as it seems that the incidence of thalassæmia is increasing in Australia. A case of thalassæmia major is described, and the clinical features of the condition are briefly stated with reference to our experience at the Adelaide Children's Hospital.

Acknowledgements.

Our thanks are due to the honorary medical staff and to all those at the Adelaide Children's Hospital who have helped in the preparation of this report, and also to Dr. E. B. Sims for permission to present the case.

References.

BAKER, S. J., and O'NEILL, J. J. (1951), "Cooley's Anæmia: A Review and Presentation of an Affected Family", MED. J. AUST., 2: 527.

CAFFEY, J. (1937), "Skeletal Changes in Chronic Hæmolytic Anæmias", *Amer. J. Roentgenol.*, 37: 293.

CAFFEY, J. (1951), "Cooley's Erythroblastic Anæmia", *Amer. J. Dis. Child.*, 45: 547.

COOLEY, T. B., and LEE, P. (1927), "A Series of Cases of Splenomegaly in Children with Anæmia and Peculiar Bone Changes", *Trans. Amer. pediat. Soc.*, 37: 29; also published in *Amer. J. Dis. Child.*, 1925, 30: 447.

COOLEY, T. B. (1932), "Erythroblastic Anæmia", *Amer. J. Dis. Child.*, 43: 705.

NEWMAN, G. G. H. (1959), "Intraperitoneal Transfusion of Blood in a Child with Cooley's Anæmia", *Lancet*, 1: 230.

SINN, H. J. (1942), "Cooley's Anæmia: Report of a Case," MED. J. AUST., 1: 579.

SMITH, C. M. (1943), "Familial Blood Status in Cases of Mediterranean Anæmia", *Amer. J. Dis. Child.*, 65: 681.

"SELF-STERILIZATION" OF CHEMICALLY TREATED BLANKETS.

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IT is unfortunate that a clear distinction has not been drawn between the immediate and prolonged effects of chemical sterilization of blankets. A number of highly active antibacterial substances have been suggested; for example, Rountree (1946) used an oil-water emulsion with cetyl pyridinium bromide ("Fixanol C"), Blowers and Wallace (1955) recommended cetyl trimethylamine bromide ("Cirrasol O.D.") and Hudson *et alii* (1959) studied the effect of "Permachem" treatment. These authors have been cautious in making definitive statements that the treatments which they have recommended will produce a self-sterilizing textile. However, Barnard (1952) claimed that after impregnation with "Fixanol C", blankets to a large extent remained self-sterilizing. Later, Frisby (1957) showed that blankets could be kept bacteriologically clean by repeated "Cirrasol" treatments—that is, after every patient—a condition not easily met in most hospital laundries.

Quite recently there has been a number of positive claims from commercial and other sources which imply that certain chemical treatments of blankets will engender prolonged sterility during use. In *The Australian National Dry Cleaner* of October, 1959, it was stated that ". . . small scale tests had previously indicated that Hibitane salts have an affinity for the fibres of cellulose materials which render them persistently self-sterilizing". Another article in *Hospital Administration* (for the Chief Departmental Officers of every hospital in Australia) of November, 1959, claimed that ". . . pathological tests failed to find any traces of *Staphylococcus aureus* four to eight weeks after blankets had been rinsed with Hibitane". This paper did not make it clear whether the blankets were used in the wards or stored in cupboards. A similar comment was also made in *The National Hospital* (official journal of the Australian Hospital Association) of December, 1959. The continued repetition of such statements implying self-sterilization of chemically treated blankets has prompted us to report our findings on this subject.

A number of used and well-rinsed woollen blankets consisting of 30% cellulose and 70% wool fibres were treated with chemicals in a manner recommended by various authors ("Permachem"-treated blankets were processed for us in the United States and the "Hibitane" rinse was used at 0·1% concentration of the drug). The antibacterial activity of the treated blankets was tested prior to their use in the wards. This was done in two ways: first, by demonstrating a zone of inhibition of growth around a test square of blanket on a moistened surface of agar lawn culture of *Staph. aureus*; secondly, by contaminating a blanket strip with the same organism and culturing it immediately in two different fluid media. The results of these tests (Table I) leave no doubt that

the blankets contained an appreciable amount of diffusible antibacterial agents whose bacteriostatic activity could be readily reversed (or antagonized) by lubrol-lecithin media. It may be added that if dry agar plates are used in the zonal inhibition tests, much smaller zones than those recorded here are obtained.

The second stage of the experiment consisted in attaching one-foot squares of the treated blankets to freshly-laundered untreated blankets, and placing them on the beds of patients. After three days' use in the ward, the upper surfaces of the treated squares and untreated blankets were sampled by the contact plate method of Rubbo and Dixson (1960), and by a modified percussion method also described by these authors. The medium used for contact plate cultures was meat-infusion agar containing 1% lubrol W and 0·5% lecithin (the additives neutralize any carry-over of chlorhexidine ("Hibitane"), "Fixanol C", "Cirrasol O.D." or "Permachem"), and blood-agar medium was used for sampling by the percussion method. In addition, strips of treated and untreated used blankets were cultured, as before, in broth and lubrol-lecithin broth.

TABLE I.

Bacteriostatic Activity of Chemically Treated Blankets. Organism: Staphylococcus Aureus.

Blanket Treated with	Zone of Inhibition. (Agar Lawn Culture.)	Inhibition of Growth.[1]	
		Broth.	Lubrol-Lecithin Broth.
Hibitane (1 : 1000) ..	2 mm.	–	++
"Fixanol C"	4 mm.	–	++
"Cirrasol O.D."	4 mm.	–	++
"Permachem"	15 mm.	–	++
Control (untreated) ..	0	++	++

[1] Plus signs indicate degree of growth; minus signs indicate no visible growth.

A typical result of contact plating is shown in Figure I. It will be seen that the degree of contamination of the treated blankets was indistinguishable from that of the normal controls.

Table II shows the results of culturing these blankets in media with and without antagonists to the chemicals being tested. It will be seen that the degree of bacterial contamination of the treated and untreated blankets is approximately the same when the lubrol-lecithin medium is used. However, in normal broth the carry-over of chemical in the treated blanket strips was sufficient to inhibit growth of natural contaminants, thus giving a false impression of sterility. Similarly, it was possible to show the same type of bacteriostasis by the zonal inhibition test with a mixed broth culture of "ward" organisms. It is evident that the results with the media containing the chemical antagonists do not support the view that chemically sterilized blankets possess the unique property of remaining sterile during use.

It should be emphasized that these experiments were made with blankets which had been submitted to one chemical treatment. It might be suggested, as Frisby has done, that frequent treatments (after every patient) will eventually lower the bacterial counts on blankets, presumably through the build-up of a bactericidal concentration of the chemical. On the other hand, we have shown that the treated blankets used in this study possessed marked bacteriostatic properties before and after use, and it would seem unlikely that the blankets, or the patients who used them, could tolerate higher concentrations than those that were obtained in these experiments.

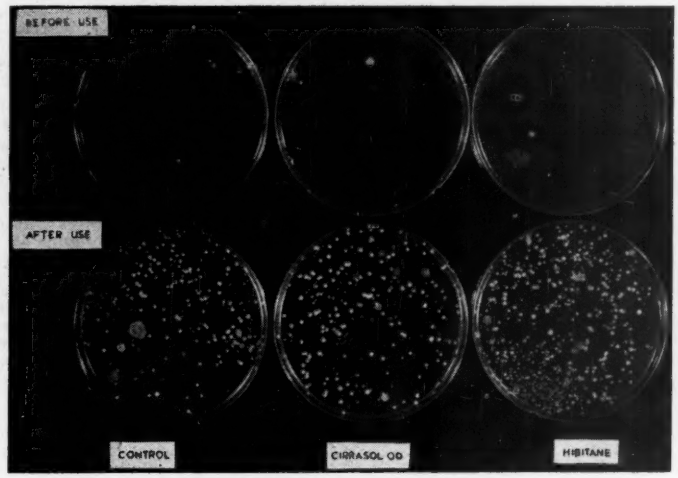

FIGURE I.

Contact plate cultures from blankets before and after three days' use in wards.

TABLE II.

Contamination of Blankets after Three Days' Use in Wards.

Blanket Treated with	Zone of Inhibition. (Agar Lawn Culture.)[1]	Culture of Blanket Strip.[2]		Sample of Blanket.[2]	
		Broth.	Lubrol-Lecithin Broth.	Contact.	Percussion.
"Hibitane" (1 : 1000)	2 mm.	–	++	++	++++
"Fixanol C"	4 mm.	–	++	++	++++
"Cirrasol O.D."	4 mm.	–	++	+++	++++
"Permachem"	20 mm.	–	++	+++	++++
Control (untreated)	0	++	++	+++	++++

[1] Mixed broth culture from a blanket.

[2] Plus signs indicate degree of growth; minus signs indicate no visible growth.

It would appear that the reason for the failure of a chemically sterilized blanket to remain sterile during use is not the inadequate concentration of antibacterial agent, but the mechanism of natural contamination. We have shown elsewhere (Rubbo, Pressley, Stratford and Dixson, 1960) that blankets are mainly contaminated by air-borne organisms travelling as free particles or on minute "fibre nuclei". Thus, in the absence of an aqueous vehicle, it is impossible for a chemical adsorbed on a woollen fibre to establish effective contact with an air-borne organism which may settle on it. If the organisms are delivered to the treated surface in an aqueous vehicle, as in Barnard's experiments, then self-disinfection may occur, but salivary droplets play a relatively unimportant role in contaminating a blanket during its use.

In conclusion, we wish to emphasize that these experiments were not designed to disprove the efficiency of chemical treatment of blankets as a method of sterilization, but merely to draw attention to the fact that the treated article will not remain sterile during use.

References.

BARNARD, H. F. (1952), "The Sterilization of Woollen Fabrics", *Brit. med. J.*, 1: 21.

BLOWERS, R., and WALLACE, K. R. (1955), "The Sterilization of Blankets with Cetyl Trimethylamine Bromide", *Lancet*, 1: 1250.

FRISBY, B. R. (1957), "Cleansing of Hospital Blankets", *Brit. med. J.*, 2: 506.

HUDSON, P. B., SANGER, G., and SPROUL, E. E. (1959), "Effective System of Bactericidal Conditioning for Hospitals", *J. Amer. med. Ass.*, 169: 1549.

ROUNTREE, P. (1946), "The Treatment of Hospital Blankets with Oil Emulsions and the Bactericidal Action of 'Fixanol C' (Cetyl Pyridinium Bromide)", MED. J. AUST., 1: 539.

RUBBO, S. D., and DIXSON, S. (1960), "A Contact Plate Technique for Determining Bacterial Contamination of Fabrics", *Lancet*, in the press.

RUBBO, S. D., PRESSLEY, T. A., STRATFORD, B. C., and DIXSON, S. (1960), "Vehicles of Transmission of Airborne Bacteria in Hospital Wards", *Lancet*, in the press.

AGORAPHOBIA: A REVIEW OF TEN CASES.[1]

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AGORA is the Greek word for market place and agoraphobia has come to mean fear of going outside, especially in crowds. It is an example of the group of symptoms referred to as phobias. Ross defines a phobia as "a specific fear which the patient knows is ridiculous but which he cannot overcome". Noyes says it is "unconscious transfer of fear from an unconscious source to a conscious and apparently unrelated source".

Phobias in obsessive-compulsive neurosis frequently take the form of compulsion to do some trivial action for fear of what may happen if it is not done. Other features of the personality type are present.

Phobias in anxiety reflect the associated lack of confidence, so that an anxious person may be afraid to go out and meet people. Other symptoms of anxiety will be present.

Phobias are not usually described as features of depression, except in so far as depression may complicate anxiety and the phobia may itself produce depression.

Phobias as isolated symptoms are written up at length in textbooks. Some early traumatic experience is repressed and emerges as a phobia. When the experience is uncovered and its relationship to the symptom revealed, the patient is cured.

Phobias are not often described in hysteria; but then practically any symptom may be hysterical and as such will have a discoverable motive.

[1] Read at a meeting of the Western Australian Branch of the Australian Association of Psychiatrists on December 10, 1959.

Since 1927, some 21 papers on agoraphobia have been indexed. Nine of these report psychoanalysis of individual patients and six deal with the condition in organic diseases. Only four references appear in the last ten years.

Source of Material.

The Havelock Clinic, a psychiatric out-patient clinic staffed by the Mental Health Services in Western Australia, has been operating for two and three-quarter years. In that time I have seen 430 patients; 10 of these volunteered agoraphobia as one of their leading symptoms (2·3%).

Digest of Data.

The following is a brief résumé of these 10 clinical histories.

Mrs. A, aged 38 years, a quarter-caste Dutch woman, had lived a life of luxury in Java. Her mother had a very dominant personality. Her husband was killed soon after war broke out, but she had one son by him. She went to Holland after the war, married again, and migrated to Australia with her husband and two daughters, leaving her son with her mother. In Western Australia she had limited money and a dull life, and wished to return to Holland. Her husband disliked crowded Holland, and admired his mother-in-law but firmly refused to live with her. The patient needed a symptom to bring pressure to bear on her husband. A healthy fear of going out during the Japanese occupation influenced her choice. She was given a variety of treatments, but clung to her symptom.

Mrs. B, aged 33 years, an intense, hysterical woman, had all her life used symptoms to command sympathy and obtain concessions from her family. She had a "marvellous" husband, who would arrive home from shift work at 11 p.m. and set to and scrub the kitchen floor if she had been too "sick" to do so during the day. She said: "I play up on them dreadfully, I'll admit it." Agoraphobia was only one of her symptoms. She had had all sorts of drugs and electro-convulsive therapy. After the first two sessions of another course she went to the country for a holiday.

Mrs. C, aged 34 years, came of an unstable family; two sisters had attempted suicide and her mother was subject to hysterical fugues. Her trouble started when she moved her clothes-line so as to catch the wind better. Her neighbour complained that it spoilt the view from her back veranda, so hosed the patient's pigeons. This had no result, so she complained to the State Housing Commission, whose inspector dismissed the complaint. The neighbour then told the Child Welfare Department that the patient's children were neglected. An inspector found the allegation unfounded. The children were next reported as wandering the streets all night and the police sent out an inspector, who took their footprints. The neighbour finally said that the patient's washing was dirty—a particularly deadly insult in lower middle-class circles. In the patient's own words, "All this made me very upset"; she reacted with fugues, agoraphobia, vomiting, headaches and belching. She dreamed of lying on a cloud and falling towards a fence of laughing faces, all of which were that of her neighbour. She was helped along with judicious sympathy until the neighbour became ill and had to have an operation. The patient then made a rapid recovery.

Mrs. D, aged 33 years, was an unwilling migrant from Holland with her husband. For all of six years in Western Australia she had wanted to return and was self-conscious about her English and afraid of saying the wrong thing. Hence she was afraid of going out and meeting people and so unhappy in Australia, and so should be taken back to Holland.

Mrs. E, aged 37 years, was an epileptic, who understandably enough disliked going out because of her fits.

Mrs. F., aged 33 years, had a dominant mother, who used symptoms to control her family. She had married a technically-minded man absorbed in mechanics, photography, etc., and was neglected and needing sympathy. She developed a fear of crowds, either in enclosed spaces or outside, and at times had to be rushed home half way through a cinema programme. She used her symptom to control the family outings.

Mrs. G, aged 28 years, had lost her father when very young, and her domineering mother lived with the patient, her husband and her only child. She had a variety of neurotic symptoms—afraid to go out, afraid to stay at

home, afraid of going mad, afraid she would kill herself, afraid she would injure her mother. She would become quite well when staying with an aunt, who told her it was all put on.

Mrs. H, aged 45 years, had lived in the city until she married a farmer rather late in life. Life was lonely and dull, and one day she was frightened by a snake. She developed a fear of going out alone, which resulted in several trips to the local township to see the doctor, as well as to the city.

Mrs. I, aged 32 years, was depressed after an alimentary disorder and the death of a child. She made a good recovery with electro-convulsive therapy.

Mrs. J, aged 55 years, had lived in a very hot part of Western Australia for 10 years and hated it, but her husband liked it. Four years ago she fell and struck her head. Full investigation gave negative results, but she became afraid to go out. She has spent the last four summers in Perth.

Analysis of Data.

The patients were all married women living with their husbands. Their ages ranged from 28 to 55 years, three-quarters being in their thirties. The average duration of symptoms was four years, yet only three had previously sought medical advice. This indicates the real severity of the symptom. When the symptom served a useful purpose, it was refractory to treatment. The possessive mother features in several histories. Dominant dames have diffident daughters.

Summary.

Of 10 consecutive cases of agoraphobia, one was an understandable reaction to epilepsy, one occurred in a depression, and one occurred in an anxiety state. In the remaining seven cases the symptom was obviously personally useful to the patients.

Acknowledgement.

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A COMPARISON OF THE DIETS OF TWO GROUPS OF SCHOOL-CHILDREN.[1]

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A PAPER was presented at the 1958 A.N.Z.A.A.S. Congress on some dietary findings of the child growth study, and later published in this Journal (Cahn and Neal, 1959). Discussion on the paper at the meeting suggested the desirability of conducting a similar type of dietary survey on children who had not attended the clinic, the aim being to determine whether or not the dietary advice given over the past five years to the study group had in any way influenced their dietary pattern or nutrient intake. The new group would then serve as a form of control for the study. Such a survey was conducted in 1959, in addition to the usual investigations for the study group. For the latter this consists of a biannual dietary assessment, and the findings of the first assessments for 1959 are shown in the results, for comparison with those of the new or control group.

The control group consisted of school-children of comparable average age and living in similar residential areas to those of the study group. With the approval of the Education Department, the headmasters of schools in these areas were approached in order to obtain the names of parents willing to cooperate in the survey. The children were aged between six and a half and seven and a half years at the time when the assessments were made. It was not possible to investigate as thoroughly the social, economic and other conditions of this group as had been done originally for the study group, but each mother was interviewed in her home. The purpose of the interview was mainly to explain the purpose of the survey and the details of the information required on the records to be completed and returned for assessment. As for the study group, each mother kept a record of the food consumed by the child for a week, and completed a *questionnaire* giving additional information to increase the accuracy of the assessment.

It will be appreciated that each is then a somewhat selected group, in that a survey of this type can be made only when parents are willing to cooperate, which may suggest that they are more than usually interested in the children's diets.

The assessments were made by calculation of the average daily intake of certain foods or food groups. Then by the use of food tables (Osmond and Wilson, 1954), the average daily nutrient intake was calculated. The result was then compared with the Australian standards of recommended levels of intake (Wilson, 1954) for the particular nutrients. It is appreciated that the inherent errors in such a method are very considerable, and that an intake below the recommended level may not have any importance or be considered to cause effects on nutritional status or health unless this effect is confirmed by clinical, biochemical or other investigations.

In the case of the study group, the major work (Roche and Sunderland, 1959) is an investigation of physical development throughout the growth period of the children, and the dietary assessments may prove to have significance when all results are correlated. When the assessment of the diet is made, other features are noted, such as the type of meal pattern, abnormalities in respect to the consumption of particular types of foods or food groups such as sugars and starches, or any factor which may influence nutritional state.

Results.

The results are shown as frequency-distribution tables of intake of selected nutrients for the girls, the boys and the total, for both study and control groups. The standards are indicated by the line marked "Standard" and it should be noted that these apply for both sexes from the ages of five to eight years. The mean intake for each nutrient has also been calculated for each group and each subdivision of the group. The tables refer to 147 school-children aged from 6·5 to 7·5 years.

The nutrient intakes compared are for calories, protein, vitamin A and carotene, thiamine, riboflavin and ascorbic acid. Each of these will be considered separately. First, with regard to calories, it will be noted from Table I that for the study group girls the mean intake is lower than for the controls. This may mean that our advice concerning restriction of highly refined carbohydrate foods has been taken, or it may result from the fact that the group is larger and contains some girls with abnormally low intakes. In all cases the means are well above recommended levels, and there appears little difference from the boys or the totals for the groups.

There does not appear to be any difference in the results for protein (see Table II), and here also the means are well above recommended levels and the scatter is not wide; hence this is a reasonably consistent finding.

The means for calcium, as shown in Table III, are at the recommended level. Thus, for approximately half the children, calcium intake is below the standard, which is consistent with findings for other surveys. It does not appear then that our efforts to increase intake for the study group have been particularly successful, as the means are all at reasonably similar figures.

The findings for vitamin A and carotene in Table IV do show some differences, particularly in respect to the

[1] Read at the A.N.Z.A.A.S. Congress, Perth, August, 1959.

boys' diets. However, the means are all considerably above the recommended levels, a result which has been found consistently throughout the years for the study group, and no efforts have been made to suggest altering the level.

TABLE I.
Calories: Frequency.

Calories: Intervals.	Child Growth Study Group.			New Group—Control.		
	Girls.	Boys.	Total.	Girls.	Boys.	Total.
3000 or over ..	—	1	1	—	—	—
2750 to 3000 ..	—	—	—	—	1	1
2650 to 2700 ..	—	—	—	—	1	1
2550 to 2599 ..	—	—	—	—	—	—
2500 to 2549 ..	2	1	3	—	—	—
2450 to 2499 ..	—	2	2	—	—	—
2400 to 2449 ..	—	1	1	—	1	1
2350 to 2399 ..	1	—	1	—	—	—
2300 to 2349 ..	1	—	1	1	—	1
2250 to 2299 ..	1	3	4	1	2	3
2200 to 2249 ..	—	2	2	2	2	4
2150 to 2199 ..	2	1	3	—	2	2
2100 to 2149 ..	5	4	9	1	4	5
2050 to 2099 ..	3	1	4	2	1	3
2000 to 2049 ..	2	3	5	1	2	3
1950 to 1999 ..	3	1	4	2	2	4
1900 to 1949 ..	4	5	9	—	3	3
1850 to 1899 ..	2	5	7	1	2	3
1800 to 1849 ..	3	3	6	—	2	2
1750 to 1799 ..	3	2	5	1	—	1
1700 to 1749 ..	5	2	7	2	1	3
Standard[1]						
1650 to 1699 ..	6	2	8	3	3	6
1600 to 1649 ..	2	1	3	—	—	—
1550 to 1599 ..	2	—	2	—	—	—
1500 to 1549 ..	—	1	1	—	—	—
1450 to 1499 ..	—	1	1	2	—	2
1400 to 1449 ..	2	1	3	—	1	1
1350 to 1399 ..	1	—	1	—	—	—
1300 to 1349 ..	2	—	2	—	—	—
1250 to 1299 ..	—	—	—	—	—	—
1200 to 1249 ..	1	—	1	—	—	—
1000 to 1049 ..	—	—	—	—	—	—
850 to 899 ..	1	—	1	—	—	—
Total	55	43	98	19	30	49
Mean Calorie intake	1834	2009	1918	1911	2036	1987

[1] Standard, five to eight years, all groups, both sexes.

TABLE II.
Protein Intake: Frequency.

Protein (Grammes): Intervals.	Study Group.			New Group.		
	Girls.	Boys.	Total.	Girls.	Boys.	Total.
120 or over ..	—	1	1	—	—	—
85 to 89·9 ..	3	3	6	—	—	—
80 to 84·9 ..	2	3	5	—	—	—
75 to 79·9 ..	5	1	6	1	7	8
70 to 74·9 ..	4	5	9	2	4	6
65 to 69·9 ..	8	7	15	1	4	5
60 to 64·9 ..	10	9	19	5	7	12
55 to 59·9 ..	10	3	13	4	4	8
Standard[1]						
50 to 54·9 ..	4	9	13	1	1	2
45 to 49·9 ..	5	1	6	5	2	7
40 to 44·9 ..	4	1	5	—	1	1
Total	55	43	98	19	30	49
Mean intake ..	62·4	66·2	64·1	58·8	65·2	62·7

[1] See footnote, Table I.

Thiamine intake, (Table V), like calcium, shows the common finding for Australian diets with the means all below the standards. If this intake was related to total calorie intake, the results would read as follows: for each 1000 Calories, the mean thiamine intake for study-group girls is 0·485 mg. and for the boys 0·431 mg.; for the control group, it is 0·457 mg. for girls, and 0·424 mg. for boys. These means are all above the suggested minimal range of 0·2 to 0·3 mg. per 1000 Calories, but some individual intakes fail to reach this level.

TABLE III.
Calcium Intake: Frequency.

Calcium (Grammes): Intervals.	Study Group.			New Group.		
	Girls.	Boys.	Total.	Girls.	Boys.	Total.
20·0 or over ..	—	1	1	—	—	—
1·9 to 1·99 ..	1	—	1	1	—	1
1·8 to 1·89 ..	2	—	2	—	—	—
1·7 to 1·79 ..	—	—	—	—	1	1
1·6 to 1·69 ..	2	1	3	—	1	1
1·5 to 1·59 ..	—	1	1	1	1	2
1·4 to 1·49 ..	—	8	8	—	—	—
1·3 to 1·39 ..	4	1	5	1	3	4
1·2 to 1·29 ..	3	5	8	1	3	4
1·1 to 1·19 ..	10	3	13	3	4	7
1·0 to 1·09 ..	9	2	11	1	4	5
Standard[1]						
0·9 to 0·99 ..	9	7	16	3	6	9
0·8 to 0·89 ..	7	8	15	1	1	2
0·7 to 0·79 ..	6	2	8	3	1	4
0·6 to 0·69 ..	2	3	5	4	3	7
0·5 to 0·59 ..	—	1	1	—	—	—
0·4 to 0·49 ..	—	—	—	—	2	2
Total	55	43	98	19	30	49
Mean intake ..	1·04	1·06	1·05	0·99	1·16	1·09

[1] See footnote, Table I.

The means for riboflavin, shown in Table VI, are all above the standard figure, with a difference which may prove to be significant between the controls and the study group. We do stress to the mothers the value of milk as a foodstuff, but as calcium intake shows no difference between groups it would not appear that milk is implicated, and the variations may be due to differ-

TABLE IV.
Vitamin A and Carotene Intake: Frequency.

Vitamin A and Carotene: Intervals.	Study Group.			New Group.		
	Girls.	Boys.	Total.	Girls	Boys.	Girls.
10,000	—	1	1	—	—	—
7500 to 7750 ..	1	1	1	—	—	—
7000 to 7250 ..	1	2	3	—	—	—
6500 to 6749 ..	4	—	4	—	—	—
6250 to 6499 ..	1	1	2	—	2	2
6000 to 6249 ..	3	1	4	—	—	—
5750 to 5999 ..	7	2	9	1	1	2
5500 to 5749 ..	4	6	10	1	4	5
5250 to 5499 ..	2	5	7	—	3	3
5000 to 5249 ..	4	5	9	1	5	6
4750 to 4999 ..	2	4	6	2	2	4
4500 to 4749 ..	4	3	7	3	4	7
4250 to 4499 ..	4	4	8	2	2	4
4000 to 4249 ..	3	1	4	2	2	4
3750 to 3999 ..	7	1	8	2	1	3
3500 to 3749 ..	1	1	2	2	2	4
3250 to 3499 ..	2	2	4	—	—	—
3000 to 3249 ..	2	1	3	2	1	3
Standard[1]						
2750 to 2999 ..	1	1	2	—	—	—
2500 to 2749 ..	1	—	1	—	—	—
2250 to 2299 ..	1	1	2	—	—	—
Total	55	43	98	19	30	49
Mean intake ..	4889	5186	5020	4771	4796	4787

[1] See footnote, Table I.

ences in consumption of yeast extracts which have a high riboflavin content.

Ascorbic acid (Table VII), is the one nutrient which shows a marked difference between the groups, the mean

TABLE V.
Thiamine Intake: Frequency.

Thiamine (Milligrammes): Intervals.	Study Group.			New Group.		
	Girls.	Boys.	Total.	Girls.	Boys.	Total.
1·8 or over ..	1	1	2	—	—	—
1·7 to 1·79 ..	—	1	1	1	—	1
1·6 to 1·69 ..	1	—	1	—	—	—
1·5 to 1·59 ..	—	—	—	1	—	1
1·4 to 1·49 ..	2	—	2	—	2	2
1·3 to 1·39 ..	1	—	1	1	—	1
1·2 to 1·29 ..	2	2	4	—	1	1
1·1 to 1·19 ..	7	—	7	—	2	2
1·0 to 1·09 ..	4	2	6	3	3	6
0·9 to 0·99 ..	5	6	11	2	2	4
Standard[1]						
0·8 to 0·89 ..	8	13	21	1	6	7
0·7 to 0·79 ..	13	9	21	4	7	11
0·6 to 0·69 ..	6	8	14	3	5	8
0·5 to 0·59 ..	3	—	3	3	2	5
0·4 to 0·49 ..	2	1	3	—	—	—
Total	55	43	98	19	30	49
Mean intake ..	0·89	0·86	0·878	0·875	0·86	0·868

[1] See footnote, Table I.

intake in the study group being lower, although in all cases means are well above recommended levels. Of the total of 147 children only 12 have an intake less than 30 mg., which is the recommended level. This difference between groups is hard to explain, and the

TABLE VI.
Riboflavin Intake: Frequency.

Riboflavin (Milligrammes): Intervals.	Study Group.			New Group.		
	Girls.	Boys.	Total.	Girls.	Boys.	Total.
4·25 or over ..	1	—	1	—	—	—
4·0 to 4·249 ..	—	—	—	—	—	—
3·75 to 3·99 ..	—	—	—	—	1	1
3·5 to 3·749 ..	—	1	1	—	—	—
3·25 to 3·49 ..	—	—	—	—	—	—
3·0 to 3·249 ..	1	1	2	—	—	—
2·75 to 2·99 ..	1	1	2	—	—	—
2·5 to 2·749 ..	3	2	5	—	2	2
2·25 to 2·49 ..	6	7	13	1	3	4
2·0 to 2·249 ..	10	7	17	4	6	10
1·75 to 1·99 ..	5	10	15	2	4	6
1·5 to 1·749 ..	21	7	28	5	8	13
1·25 to 1·49 ..	4	4	8	2	—	2
Standard[1]						
1·0 to 1·249 ..	2	3	5	5	6	11
1·0 or under ..	1	—	1	—	—	—
Total	55	43	98	19	30	49
Mean intake ..	1·90	1·92	1·91	1·63	1·85	1·76
Standard 1·3 milligrammes.						

[1] See footnote, Table I.

opportunity for nutritional education appears from these figures to have had an adverse effect, although in only a few cases has ascorbic acid intake been discussed with the mothers.

Generally, it appears that as only slight differences can be observed between the groups, the influence of the dietary advice for the study group has not resulted in an alteration of dietary pattern to any important extent.

The relative intake of highly refined carbohydrate foods has always been noted when the study groups diets are assessed, and this has also been recorded for the control group.

In Table VIII are some results obtained for 59 of the study group and the control group of 49. The amount of money spent per week is included on the *questionnaire*, and it could be assumed that this may be under-estimated. Summing up the results as submitted, it will be seen that the average spending is reasonable, and again, our advice does not appear to have had any effect. The boys appear the worse offenders in regard to the amount of money spent on sweets.

TABLE VII.
Ascorbic Acid Intake: Frequency.

Ascorbic Acid (Milligrammes): Intervals.	Study Group.			New Group.		
	Girls.	Boys.	Total.	Girls.	Boys.	Total.
199	—	1	1	—	—	—
120 to 129·9 ..	—	2	2	—	—	—
110 to 119·9 ..	—	1	1	1	1	2
100 to 109·9 ..	1	1	2	1	1	2
90 to 99·9 ..	3	1	4	2	2	4
80 to 89·9 ..	2	2	4	1	2	3
70 to 79·9 ..	3	3	6	2	5	7
60 to 69·9 ..	11	8	19	4	7	11
50 to 59·9 ..	7	7	14	3	4	7
40 to 49·9 ..	14	13	27	3	3	6
30 to 39·9 ..	8	2	10	1	3	4
Standard[1]						
20 to 29·9 ..	4	2	6	1	2	3
Under 20 ..	2	1	3	—	—	—
Total	55	43	98	19	30	49
Mean intake ..	50·8	61·6	55·5	65·8	63·8	64·6

[1] See footnote, Table I.

The calories derived from sugars and highly refined starchy foods lacking in other nutrients, particularly thiamine, are shown for each group in Table IX.

The means are in the range of 300 Calories daily, representing approximately one-sixth of total calorie intake, and in the upper levels between one-quarter and one-third of total calories, an amount which must be regarded as excessive.

It was also thought that an analysis of the breakfast and school-lunch patterns might be of interest. The

TABLE VIII.
Weekly Expenditure on Sweets.

Amount Spent per Week.	Study Group.		New Group.	
	Girls (22).	Boys (37).	Girls (19).	Boys (30).
Up to 3d. ..	8	11	9	11
6d. to 1s. ..	9	8	6	9
1s. to 1s. 6d. ..	4	9	2	7
1s. 6d. to 2s. ..	—	1	—	2
2s. to 2s. 6d. ..	1	7	2	—
2s. 6d. or over ..	—	1 (5s.)	—	1 (3s.)

majority of children consumed some form of milk beverage at breakfast, but otherwise there was great variation in meal pattern from day to day between individual children, and for each child. On the assumption that the breakfast contained a milk beverage, breakfasts were then graded as follows: (1) bread or toast only, with butter and some form of spread—jam, honey or yeast extract; (ii) precooked breakfast cereal, with or without bread, etc.; (iii) porridge with or without bread, etc.; (iv) a cooked breakfast including eggs or some other protein-rich food.

The results show that three only of the study group and four only of the controls consumed breakfast type (i) four or more times per week. Seventeen only of the study group, and 11 of the controls had type (iv) breakfast more than four times a week, while four of the study group and nine of the controls had breakfast type (iii). The balance, and the majority, consumed breakfast type (ii), which was also the usual alternative for those taking the other types of breakfasts four or more times per week.

In regard to school lunches, for the study group of 59 assessed, only three consumed regularly very protein-poor lunches, and 16 had lunches unbalanced in other respects, mainly lacking fruit—which, however, was usually consumed at other times. Of the 49 control diets, five were poor in protein and eight unbalanced, again lacking fruit. It can be assumed that, in respect to intake of important nutrients, the lunches were reasonably satisfactory. However, there was a trend to excessive carbohydrate intake, as the lunches and between-meal snacks were the occasions for the consumption of biscuits, sweets, soft drinks, cake and other less desirable foodstuffs.

TABLE IX.

Daily Calories Derived from Sugars, Soft Drinks, Biscuits, Cakes.	Study Group.		New Group.	
	Girls (22).	Boys (37).	Girls (19).	Boys (30).
600 or over ..	—	1	—	—
550 to 599 ..	—	4	1	—
500 to 549 ..	1	2	—	1
450 to 499 ..	—	1	—	3
400 to 449 ..	2	3	4	4
350 to 399 ..	1	8	1	6
300 to 349 ..	7	2	3	9
250 to 299 ..	2	6	5	1
200 to 249 ..	4	4	4	4
150 to 199 ..	4	1	1	1
100 to 149 ..	1	3	—	1
50 to 99 ..	—	2	—	—
Mean intake ..	286·5	342	316·5	337·5

Summary.

The nutrient intakes and meal patterns of 98 children of the Child Growth Study were very little different from those of 49 school-children of comparable age and residential areas who had not received the dietary advice given over the past five years to the study group.

It can then be reasonably assumed that the dietary advice will not affect the work of the major study of the physical growth of normal Australian children.

Acknowledgements.

The authors wish to acknowledge gratefully the assistance and cooperation of Dr. A. Roche, Department of Anatomy, University of Melbourne, Director of the Child Growth Study, who has made it possible for us to conduct the dietary surveys and also to present the findings as a separate report, and also the staff of the Growth Clinic for their assistance.

References.

CAHN, A., and NEAL, K. (1959), "Nutritional and Dietary Aspects of the Melbourne Child Growth Study", MED. J. AUST., 2: 549.

OSMOND, A., and WILSON, W. (1954), "Tables of Composition of Australian Foods", Revised Edition, Nutritional Committee, National Health and Medical Research Council, Commonwealth Department of Health, Canberra.

WILSON, W. (1954), "Recommended Dietary Allowances for Australia", MED. J. AUST., 2: 113.

ROCHE, A. F., and SUNDERLAND, S. (1959), "Melbourne University Child Growth Study", MED. J. AUST., 1: 559.

ANTI-M ANTIBODY AS A RARE CAUSE OF HÆMOLYTIC DISEASE OF THE NEW-BORN.

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ONE of the rarer atypical antibodies occasionally found in the serum of blood donors, pregnant women or patients requiring transfusion is anti-M. This is generally a naturally occurring antibody, in that its presence does not follow any known specific antigenic stimulus, and it reacts best at room temperature, or at 5° C. We have identified and investigated not less than 20 examples of anti-M antibodies over a period of ten years. When anti-M is found in a patient's serum before blood transfusion, the appropriate blood of type N must be given. When anti-M is found in a woman's serum during pregnancy, the antibody titre is measured regularly until confinement, and should the baby's blood be of type MN, the child is observed after birth for possible evidence of hæmolytic disease of the new-born. Similar precautions are taken in all subsequent pregnancies. The present paper reports observations on a family in which three successive children of type MN showed clinical manifestations of hæmolytic disease of the new-born requiring treatment, when the only atypical antibody demonstrable in the maternal blood was anti-M.

Some examples of anti-M capable of reacting at 37° C. and a limited number of blood transfusion reactions attributed to anti-M have been reported. These were referred to by Freiesleben and Knudsen (1957), when they reported a third example of a human incomplete immune anti-M antibody of high titre demonstrated by the indirect Coombs test; but a search of the literature has revealed few claims that anti-M antibody may cause hæmolytic disease. Bromchil (1951) reported the first example of isoimmunization by the M antigen as a cause of hæmolytic disease of the new-born. Levine (1955), in discussing hæmolytic disease due to antibodies other than anti-D, believes that if A-B-O incompatibilities are excluded, only 1% of cases will be due to antibodies other than anti-D. At that time he listed 23 other blood factors known to be involved in cases of hæmolytic disease, and amongst these his laboratory had encountered only one example of anti-M.[1]

Jakobowicz and Bryce (1951) have reported that anti-M agglutinating antibody can be placenta-permeable, and in this respect differs from Rh agglutinating antibody, which does not pass into the infant's circulation, although Rh incomplete antibody readily does so. In the case studied by them, a baby of blood type N had an agglutinating anti-M titre of 120, equal to that in the mother's serum, and we have found equal titres in cord and maternal blood in other babies of blood type N whose mother has an anti-M antibody.

It is not easy to incriminate anti-M as the cause of any particular case of hæmolytic disease. In one family in which anti-M was initially regarded as the damaging antibody, it was shown that high-titre immune anti-A and anti-B antibodies in the maternal serum were probably the real causative factors of the disease. This case (Prest, Bonnin, Simmons and Newland, 1955) illustrated that the obvious anti-M antibodies could cause other damaging antibodies to be overlooked.

[1] Since submitting the present paper for publication we have seen the report of Stone and Marsh (1959), in which hæmolytic disease of the new-born due to anti-M antibody was demonstrated in twins, one of whom was delivered as a macerated fœtus and the other was born alive, but in a deeply jaundiced condition.

Hæmolytic Disease of the New-born in Mrs. Z.'s Family.

The following is briefly the history of hæmolytic disease in Mrs. Z.'s family.

Mr. Z. was of Group O MN Rh_1Rh_1 (CDe/CDe). Mrs. Z. was of Group A_1B N Rh_2rh (cDE/cde). Mrs. Z.'s obstetrical history was as follows.

In 1953, Mrs. Z. (then Mrs. T.) was delivered of a macerated fœtus with a knotted cord. At that time anti-M antibody was demonstrated in her serum to a titre of 16.

In 1957, Mrs. Z. was delivered of an apparently healthy, normal, full-term baby of blood group B MN. The direct Coombs test on the baby's cells produced a negative result. Three weeks after birth the baby was admitted to the Royal Children's Hospital, Melbourne, with a history of jaundice of four days' duration. On examination of the baby, there was minimal jaundice with gross pallor. The hæmoglobin value was 3·6 grammes per 100 ml. Blood examination showed mild anisocytosis and polychromasia affecting the erythrocytes, and a "shift to the left" in the neutrophils; the platelets were normal, and the proportion of reticulocytes was 3·2%. The result of the direct Coombs test was negative. The urine was normal on microscopic examination. The baby was transfused slowly with 5 oz. of type N blood. Although there was no clinical evidence of infection, the elevated leucocyte count prompted the administration of crystalline penicillin for five days. The baby was discharged from hospital with a hæmoglobin value of 14·5 grammes per 100 ml., and subsequent progress was good.

In 1958, Mrs. Z. was delivered of a baby in good clinical condition. The spleen was just palpable, and the liver could be felt one finger's breadth below the costal margin. The baby's blood group was A MN, and the result of the direct Coombs test was negative. The hæmoglobin value of the peripheral blood was 19·1 grammes per 100 ml. On the second day of life the hæmoglobin value had fallen to 17·6 grammes per 100 ml., and the baby was mildly icteric. The icterus increased, and an exchange transfusion of 485 ml. of Group AN, Rh-negative blood was given. On the fourth day of life a second exchange transfusion of 850 ml. of Group AN blood was given. The jaundice appeared to be fading, but the hæmoglobin value was only 11·2 grammes per 100 ml. on the sixth day, and dropped to 8·8 grammes per 100 ml. by the tenth day. A simple transfusion of 135 ml. of Group AN, Rh-negative blood was given, and thereafter the baby's condition remained satisfactory.

In 1959, Mrs. Z.'s fourth pregnancy was terminated by medical induction of labour at 37 weeks. The baby was in good condition, but became slightly jaundiced soon after birth. The spleen was palpable, and the liver could be palpated two fingers' breadth below the costal margin. The hæmoglobin value was 13 grammes per 100 ml. in the cord blood and 16·25 grammes per 100 in the peripheral blood. The baby's blood group was B MN, Rh-positive. The result of the direct Coombs test was negative. The baby's serum contained anti-M antibodies detectable by saline and albumin techniques at room temperature, but not detectable at 37° C. The jaundice increased, and the serum bilirubin level was 11·4 milligrammes per 100 ml. on the second day. An exchange transfusion of 660 ml. of Group BN blood was given. Two days later the jaundice had increased, and the serum bilirubin level rose from 16·9 to 21·6 milligrammes per 100 ml. on the fifth day. A later reading on the same day was 19·9 milligrammes per 100 ml., and on the following (sixth) day 19·4 milligrammes per 100 ml. The jaundice then gradually decreased, until by the eleventh day it had completely faded. The baby was well when discharged from hospital.

Discussion.

Through Mrs. Z.'s second, third and fourth pregnancies regular anti-M titrations were performed. Data summarized from the records of three laboratories showed that the anti-M titre at room temperature over a period of three years varied from approximately 8 to 32, and the antibody was sometimes demonstrable at 37° C. to a titre of about 2. During the period of testing there was no evidence that incomplete anti-M antibody was also present. In each of the three babies investigated, anti-M antibody was detected in the cord blood at a titre of from 2 to 8; this again demonstrated that agglutinating anti-M antibody is placenta-permeable. Unfortunately elution tests were not carried out on the cord cells; but as it seemed unlikely that a low-titre "cold" anti-M antibody would be the cause of hæmolytic disease in three successive babies, a number of attempts were made to detect the presence of other atypical antibodies in Mrs. Z.'s serum by the use of selected cell samples from a large panel of known blood types. Although the tests were performed by the saline, albumin, indirect Coombs, papain, and indirect Coombs on papain-treated cell techniques, all gave uniformly negative results, and no atypical antibody other than anti-M was detected at any period. After absorption of the anti-M antibodies from the maternal serum, it did not react with the husband's red cells by the saline, albumin, anti-globulin and papain methods. Thus hæmolytic disease of the new-born due to a private or family antigen was excluded. As the three babies were diagnosed as suffering from hæmolytic disease by different pædiatricians, and as the infants responded in the expected manner to blood transfusion, we have accepted that a low-titre placenta-permeable anti-M antibody must have been responsible for all three cases of hæmolytic disease, although each child's blood gave a negative response to the direct Coombs test at birth.

It is recognized that antibody titres in the maternal and cord serum do not necessarily correlate with the severity of hæmolytic disease, even when the antibody is incomplete and therefore most likely placenta-permeable. This is particularly so when the hæmolytic disease is due to A-B-O incompatibility. In most cases of hæmolytic disease of the new-born a positive reaction to the direct Coombs test is obtained, but the test is not infallible, as in our hands, and with the use of the standard reagent and technique (Commonwealth Serum Laboratories Coombs reagent), not only the present patients, but the majority of subjects of A-B-O sensitization fail to give a positive reaction. The absence of a positive reaction to the direct Coombs test on the cord cells should not lull either the pathologist or the clinician into a false sense of security. In some cases observed by us, anti-M has been detected in the cord blood of type N babies at a titre equal to that in the maternal serum, while in the three affected type MN babies discussed here, the anti-M titre in the cord serum has been only approximately one-quarter of that demonstrated in the maternal serum. As the antibody appears to be placenta-permeable, it must be assumed that fœtal antigens have been responsible for this reduction in titre, without the cells becoming "Coombs-positive". The selective process which protects a baby from the action of an antagonistic maternal antibody is not known. We can only hazard that, in the present family, either the nature of the particular M agglutinin or a quality of the fœtal cells or the absence of some protective mechanism permits a low-titre complete anti-M antibody acting best at a low temperature range to damage fœtal cells. In spite of recent advances in this field, there is still much to be learned concerning the protective mechanisms possessed by some babies against damaging antibodies.

Summary.

Three successive babies of blood type MN in one family suffered from hæmolytic disease of the new-born when the only atypical antibody demonstrable was a low-titre placenta-permeable anti-M agglutinating antibody, most active at a low temperature range.

Acknowledgement.

We thank Dr. Margaret Mann and the Royal Children's Hospital, Melbourne, for making available the clinical data on the first affected baby.

References.

BROMCHILL, G. (1951), *"Isoinmunizacion por el Antigeno M Neuva Causa de Enfermedad Hemolitica Neonatal", Rev. Soc. argent. Hemat. Hemoter., 3: 104.*

FREIESLEBEN, E., and KNUDSEN, E. E. (1957), "A Human Incomplete Immune Anti-M", P. H. Anderson, Festskrift, *Munksgaard, Copenhagen: 26.*

JAKOBOWICZ, R., and BRYCE, L. M., (1951), "A Note on a Placenta-Permeating Anti-M Agglutinin", MED. J. AUST., *1: 365.*

LEVINE, P. (1955), "Hæmolytic Disease due to Antibodies other than Anti-D", *Rev. hémat.*, 10: 215.

PREST, E., BONNIN, J. A., SIMMONS, R. T., and NEWLAND, B. T. J. (1955), "Hæmolytic Disease of the New-Born due to ABO Isosensitization in Association with Potent Anti-M Agglutinins", MED. J. AUST., 2: 153.

STONE, B., and MARSH, W. L. (1959), "Hæmolytic Disease of the Newborn Caused by Anti-M", *Brit. J. Hæmat.*, 5: 344.

WARM BLOOD FOR EXCHANGE TRANSFUSION IN INFANTS.

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DESPITE the extensive employment of exchange transfusion in the treatment of erythroblastosis fœtalis and the icterus associated with prematurity, sudden death during or shortly after the procedure still occurs too frequently. Autopsy commonly provides no conclusive reason for death, and therefore many explanations have been offered in the past.

Because these infants frequently approach transfusion in excellent condition and have an apparently hopeful prognosis, their sudden death is particularly distressing to the parents and doctor. Any factors decreasing the incidence of such a catastrophe merit careful attention.

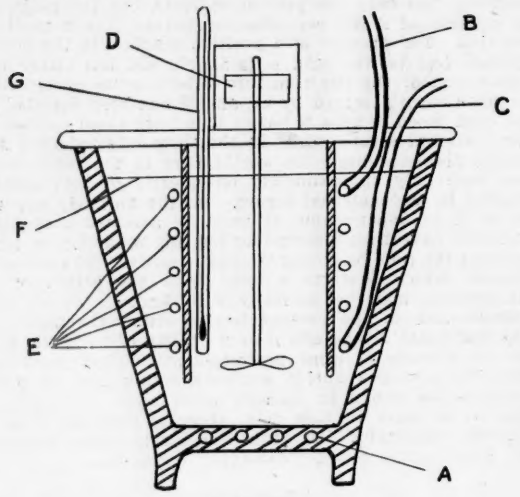

FIGURE I.

Schematic diagram of blood-warming apparatus. A, thermostatically controlled water-bath. B, cold blood inlet. C, warm blood outlet. D, electric motor driving stirrer. E, coils of plastic tubing. F, water level. G, thermometer.

The use of stored citrated blood has certain biochemical hazards, which were clearly enumerated and investigated by Farquhar and Hilton Smith in 1958. A high concentration of citrate ions may itself be toxic or, by reducing the blood level of ionized calcium, produce tetany. The use of calcium gluconate during the exchange, and care not to exceed the body's power to metabolize citrate by very fast rates of transfusion, partly overcome these hazards. In stored blood, a gradual increase in potassium ion concentration in the plasma fraction occurs; but if the blood is less than three days old, cardiac complications from hyperkalæmia should not occur (Campbell, 1955).

Careful attention to these factors alone does not prevent occasional mishaps. In 1955, Pew drew attention to the dangers associated with the use of excessively cold blood. This worker successfully treated a baby with cardiac arrest by cardiac massage. He then noted that blood injected into the umbilical catheter produced definite cooling of the right auricle, this chilling being appreciated with the operator's hand around the heart at each fresh injection of blood. Marting, Wagner and Wilson (1955) subsequently described a rather cumbersome apparatus for safely warming stored blood to a temperature acceptable to the infant, and reported facilitation of safe exchange transfusion with this apparatus. Robertson (1959), in discussing the cause of sudden death during exchange transfusion, gave the problem some consideration; but only in the article by Marting *et alii* has any apparent attempt been made to measure the temperature of blood actually reaching the infant.

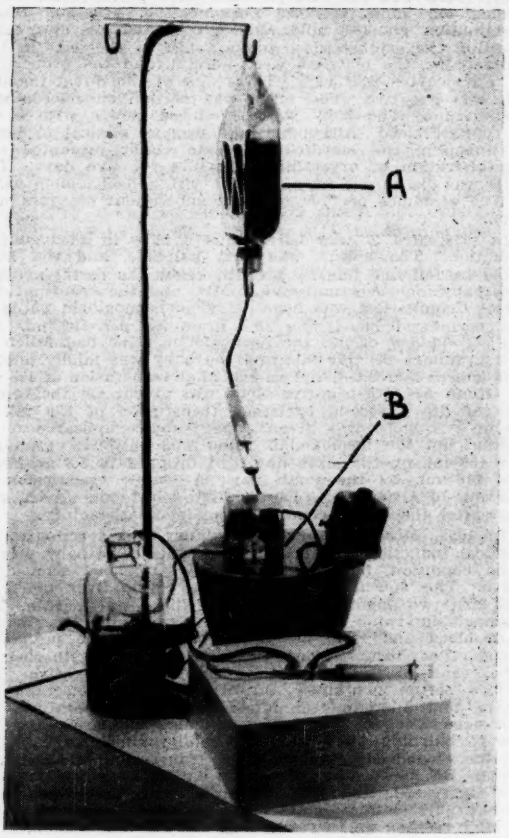

FIGURE II.

General view of exchange apparatus. A, blood. B. blood warmer.

At the Royal Women's Hospital, Melbourne, concern was felt because of the occasional loss of infants during exchange transfusions. No clear cause for death was evident from available biochemical or autopsy evidence in most cases, but on clinical grounds cold blood was incriminated. In an attempt to elucidate the problem, temperature measurements were made employing the method of blood warming then in current use in the hospital.

It was ascertained that blood left the blood-bank refrigerator on the hospital premises at about 9° C., and usually had warmed to 12° C. by the time it reached the theatre and was placed to warm in a container of water at 32° C. In the following half hour, during which time the baby was prepared for the transfusion, the bottle of blood had warmed to about 20° C., and by the conclusion

of an exchange transfusion lasting 45 minutes, the temperature still did not exceed 29° C. In a warm room, slight warming did occur in the syringe, tapset and tubing, so that under favourable conditions, blood reached the umbilicus at 21° C. at the commencement and at 29° C. at the conclusion of an exsanguination transfusion.

Theoretically, this cold blood might cause the infant's temperature to drop by 4° C., if no external heat was supplied and the temperature-regulating mechanism was functioning poorly. In practice, despite the liberal use of hot-water bottles, a temperature loss of up to 2° C.

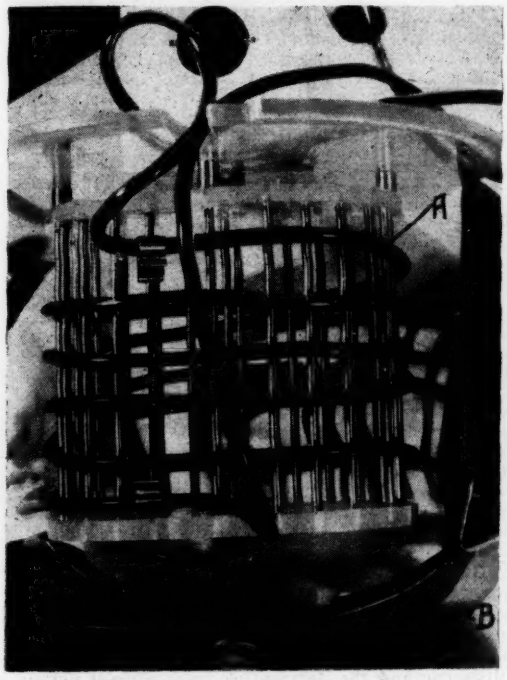

FIGURE III.

Close-up view of heating coil. A, coil of plastic tubing. B, thermostatically controlled water-bath.

was not unusual in small infants. But of even more importance than this generalized fall in body temperature was the local effect of a stream of cold blood entering the right auricle; this, by cooling the pacemaker and conduction mechanism, might cause cardiac irregularity or arrest.

A blood-warmer was therefore devised, tested in the laboratory and subsequently used in 30 consecutive exchange transfusions. No death occurred in this series, and in all but one case the desired exchange volume of 220 ml. of blood per kilogram of body weight was attained without incident. In the one exception, the exchange was abandoned solely because of difficulty in bleeding. In no case did bradycardia or cardiac irregularity appear, and no difficulty was found in maintaining body temperature constant within ± 0·2° C., as measured rectally with an electric thermometer.

The blood-warming apparatus consisted essentially of 3 metres of polyvinyl tubing immersed in a thermostatically controlled water bath. The coil of tubing was part of a sterile disposable intravenous set, supplied with extra tubing length, but standard in all other respects.[1] The water bath was an industrial glue-pot,[2] which was adjusted to maintain the contained water between 37·5° C. and 39° C. The plastic lid constructed for this glue-pot was fitted with a small electric motor driving a stirrer, and also with a plastic and steel cage upon which the intravenous tubing was coiled. A brisk circulation of water was found to be essential for efficient operation of the machine, hence the inclusion of the agitator. A thermometer also was provided.

Details of construction are shown in the accompanying diagram and photographs (*Figures I to IV*). The bottle of cold blood is elevated so that gravity aids in filling the syringe. The length of tubing from the water-bath to the tapset is kept as short as possible to decrease heat loss in this region.

It will be noted that blood is warmed just prior to its introduction into the patient, and there are none of the objections associated with the rapid warming of an entire bottle of blood. Also, it is impossible for blood to exceed the temperature of the water-bath, which is kept between 37·5° C. and 39° C.

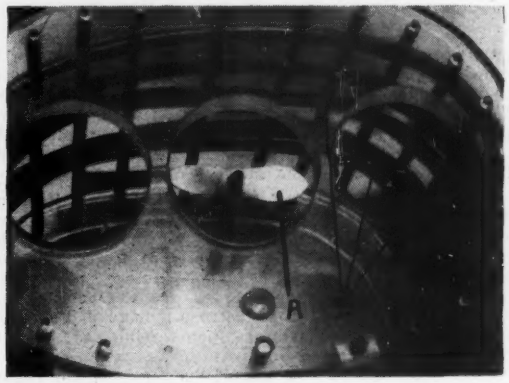

FIGURE IV.

A, stirrer; B, heating coils; shown from below.

Blood warmed in this fashion is not adversely affected. In an isolated experiment, blood held in the machine at 38·5° C. suffered no change in sodium, potassium or chloride ion concentration even after a lapse of 20 minutes.

In trials before clinical introduction of the machine, and later during its use with patients, blood was delivered into the umbilical catheter at 1·5° C. lower than the water-bath temperature. This proved entirely acceptable in clinical practice.

In the first two cases the body temperature became elevated to 38° C., but in all subsequent cases this error of overheating with hot-water bottles was not repeated. It is considered that adequately warm blood has contributed a further measure of safety to the exchange procedure, although it must be stressed that careful attention to many other technical details is of great importance also.

Summary.

The dangers associated with the use of cold blood during exchange transfusion are reviewed, and the temperature of blood reaching the baby with inadequate warming is investigated.

A simple, inexpensive and convenient blood-warmer is described and illustrated. The safety of the apparatus was established in the laboratory, and thereafter in the successful use in 30 clinical cases of exchange transfusion in the neonatal period.

References.

CAMPBELL, W. A. B. (1955), "Potassium Levels in Exchange Transfusion", *Arch. Dis. Childh.*, 30: 513.

FARQUHAR, J. W., and SMITH, H. (1958), "Clinical and Biochemical Change during Exchange Transfusion", *Arch. Dis. Childh.*, 33: 142.

[1] Supplied by Tuta Products Pty. Ltd.

[2] Supplied by Birko Electrics Pty. Ltd.

MARTING, F. L., WAGONER, S. C., and WILSON, V. C. (1955), "The Use of Warm Blood for Exchange Transfusion", *A.M.A. J. Dis. Child.*, 89: 289.

PEW, W. L. (1955), "Cardiac Arrest During Exchange Transfusion", *J. Pediat.*, 47: 645.

ROBERTSON, S. E. J. (1959), "Fatal Collapse During Exchange Transfusion", MED. J. AUST., 2: 809.

Reports of Cases.

MYOCARDIAL INFARCTION COMPLICATED BY SEVERE HÆMORRHAGE FROM A SPONTANEOUS LACERATION OF THE STOMACH (MALLORY-WEISS SYNDROME).

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WE were prompted to report the following case for the following reasons: (i) the importance of considering cardio-œsophageal lacerations as a cause of hæmatemesis of unknown origin; (ii) the difficulties associated with the treatment of a patient suffering from myocardial infarction complicated by a hæmatemesis; (iii) the rarity of reports of cases of the Mallory-Weiss syndrome.

Since the description of the condition by Mallory and Weiss in 1929, only 22 cases proven by autopsy or operation have been reported in the literature. Mallory and Weiss described four cases, all in adult males who were heavy alcohol drinkers. They stressed the importance of precedent retching or vomiting as a cause of the linear lacerations situated at the gastro-œsophageal junction in the long axis of these viscera, penetrating as far as the muscularis. The following case presents many of the features described in the original and subsequent reports.

Clinical Record.

The patient was a healthy man, aged 68 years, who was examined for the first time at his place of employment on the day of his admission to hospital. He complained of moderately severe chest pain associated with severe belching. The pain was of sudden onset, was not related to exertion and was situated behind the lower third of the sternum without any radiation. The pain was accompanied by sweating, but there was no breathlessness or collapse. When examined approximately half an hour after the onset of the pain, the patient was more concerned about the astonishingly noisy eructations than the chest pain, which was now not so severe. The patient was seen to have two large noisy vomiting attacks, there being no blood in the vomitus. The patient admitted to only two previous episodes of chest pain, both not related to exertion; on the previous day he had suffered two attacks of chest pain, similar in nature to the present, except they were not as severe and were relieved by the ingestion of "Rennies" (a proprietary antacid preparation). The chest pain was not sufficiently severe to stop him working. The only relevant history was that he had suffered from indigestion for years, which was relieved by "Rennies". Four years previously he had had a barium-meal X-ray examination which revealed no abnormality. Only three weeks prior to his present illness he had had a physical check-up, including an electrocardiogram, and was proclaimed to be very fit. The patient smoked ten cigarettes and four cigars per day and drank alcohol only on social occasions, which, however, were fairly frequent.

On his admission to hospital an hour and a half after the onset of the pain, the patient was in slight distress from the pain, and again noisy eructations were a feature. The pulse rate was 92 per minute, and was regular in time and amplitude, and the blood pressure was 160/105 mm. of mercury. The heart was not enlarged and the heart sounds were normal. There was no evidence of shock or cardiac decompensation. The abdomen was soft and there were no masses and no tenderness. The liver and spleen were not palpable. Examination of the other systems revealed no abnormality, and the urine contained no sugar or albumin, but there was a dark ring of acetone. A clinical diagnosis of myocardial infarction was made.

An electrocardiogram was taken immediately and reported on by Dr. G. E. Bauer as follows: ". . . an abnormal electrocardiogram with abnormal *ST* segments in 2, 3, aVF, deep *ST* depression in anterior chest leads. Suggests recent posterior myocardial infarction." (See Figure I.)

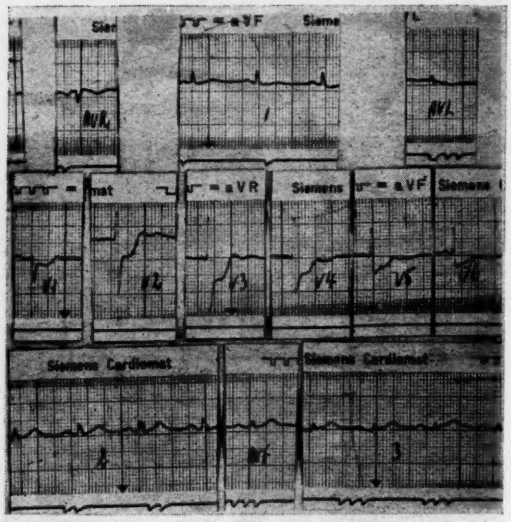

FIGURE I.
Electrocardiogram taken on the patient's admission to hospital.

Hæmatological and biochemical investigations gave the following results: the hæmoglobin value was 19·2 grammes per 100 ml.; the leucocytes numbered 13,800 per cubic millimetre, and the differential count showed normal proportions; the erythrocyte sedimentation rate was 4 mm. in the first hour. The serum glutamic oxaloacetic transaminase content was 38 units per 100 ml. (normal 0 to 40 units).

In view of the previous history suggestive of a gastric ulcer, anticoagulants were withheld, and the patient was treated with strict bed rest and light ward diet. A hypodermic injection of a quarter of a grain of morphine quickly relieved the chest pain, and the patient's eructations settled considerably. For the next six hours he felt very comfortable, and on retiring he was given a hypodermic injection of one-third of a grain of "Omnopon" more for its sedative than for its analgesic effect. Some 40 minutes later (six and a half hours after his first injection of opiate) he vomited for the first time in hospital. The vomitus amounted to 10 oz. and contained fluid and altered food. The nurse stated that the last ounce of vomitus was bright red blood. Over the next eight hours the patient vomited a further seven times, on each occasion bright red blood which clotted, and the amount totalled more than two litres.

A provisional diagnosis of (i) myocardial infarction and (ii) Mallory-Weiss syndrome or bleeding from a high gastric ulcer was made.

A blood transfusion was commenced, and a total of two litres given within the next 12 hours. Fortunately no evidence of cardiac decompensation occurred during the transfusion, and the blood pressure was maintained at 95 to 100 mm. of mercury systolic. The patient's general

condition improved sufficiently by the next morning to allow him to have a first-week ulcer diet; but two hours later he experienced a very severe attack of retrosternal chest pain and vomited brown-stained fluid (no new blood). The pain was poorly relieved by a total of two-thirds of a grain of "Omnopon" given by hypodermic injection, and the patient became rapidly shocked. This condition temporarily responded to a nor-adrenaline infusion, but finally he became stuporose and died 22 hours after his admission to hospital from what appeared to be a classical further myocardial infarction.

Biochemical investigations on the day when he died gave the following results: serum glutamic oxaloacetic transaminase content, 100 units per 100 ml.; total serum protein content, 6·9 grammes per 100 ml. (albumin 4·5 grammes, globulin 2·4 grammes per 100 ml.); direct Van den Bergh test, negative result; serum bilirubin content, 0·7 mg. per 100 ml.; thymol turbidity, 1 unit; zinc sulphate turbidity, 2 units; serum alkaline phosphatase content, 8 King-Armstrong units. A surgical consultant was acquainted of the patient's condition soon after the hæmatemesis; but it was the surgeon's opinion that in view of the patient's poor condition, immediate surgery was contraindicated.

The final clinical diagnosis was as follows: (i) recurrent myocardial infarction; (ii) arterial bleeding from a high gastric lesion (? gastric ulcer, ? Mallory-Weiss syndrome).

Autopsy Report.

The heart weighed 16 oz. There were no pericardial adhesions. Three ounces of straw-coloured fluid were present in the pericardial cavity. The heart was enlarged, owing to slight hypertrophy of the left ventricle and slight dilatation of all chambers. There was mild calcification of the base of the cusps of the aortic valve, which was otherwise normal and competent. The remainder of the valves and the endocardium were normal. There was a myocardial infarct approximately 6 by 5 cm. in area involving the anterior wall of the left ventricle, and extending from the left border to within 3 cm. of the septum and limited to the upper half of the ventricular wall. The cut surface of the myocardium showed mottled, pale-yellow, lustreless muscle flanked by a zone of congestion. The remainder of the myocardium was grossly normal. The left coronary artery showed mild atheroma in the main trunk. Its descending branch was entirely calcified and its lumen extremely narrow. The circumflex branch showed increasingly severe atheroma with calcification to a point about 3 cm. from its origin, where a calcified plaque was almost completely occluding the lumen. Attached to the distal border of this plaque was a thrombus approximately 1 cm. long. The right coronary orifice was bifid. There was notable atheroma of the larger circumflex division.

The œsophagus was normal. The stomach was dilated, but practically empty, containing thin mucus and a few small granules of altered blood. Situated along the lesser curvature, and 1 cm. below the cardio-œsophageal junction, there was a linear laceration 2·5 cm. long, with smooth, slightly pouty edges. Surrounding this tear there was an extravasation of blood into the stomach wall, where it could be seen from the mucosal surface as a dark patch approximately 10 cm. in maximum extent in the submucosa. Hæmorrhage into the lesser omentum could be seen from the external surface. In the base of the laceration a ruptured artery could be seen. Situated in the cardia on the posterior wall was a similar parallel mucosal laceration 2 cm. long. No extravasation was associated with this laceration. There was no evidence of peptic ulcer or healed ulcer (see Figure II). Situated in the distal part of the duodenum was a diverticulum 4 cm. deep and 2·5 cm. in diameter lined by a smooth congested mucosa. There was no evidence of bleeding from the diverticulum. The sigmoid colon contained numerous diverticula, from which no evidence of bleeding was apparent. There was no evidence of diverticulitis. The remainder of the intestines were normal, and there was no blood or altered blood in its contents.

The other organs were normal.

Microscopic examination of sections of the myocardium showed necrotic muscle, œdema and infiltration by polymorphs into the connective-tissue spaces and between the muscle fibres.

Microscopic examination of random sections of the stomach showed one area of normal histological appearance and another area of mild mononuclear infiltration.

FIGURE II.
Showing two lacerations situated in the cardia. The one on the left shows blood clot from a ruptured artery at its distal end.

Discussion.

A total of 23 proven cases of Mallory-Weiss syndrome have been recorded since 1929, 17 of these cases in the past seven years, there being a gap of twenty years between the second and third reports. The most important feature of these cases are set out in Table I.

The disease is predominately one affecting males, only three cases having occurred in women in the series reviewed.

There is a fairly wide age span, from 21 to 83 years; but the greatest incidence occurs between the ages of 50 and 80 years (14 cases).

In the original four cases and in the subsequent two cases reported by Weiss and Mallory (1932), the importance of alcohol as a causative factor was stressed. However, in subsequent cases, chronic alcoholism was not such a feature, for only a further four and possibly five patients were described as being heavy drinkers.

TABLE I.

Authors.	Patient's Sex and Age. (Years.)	Alcohol Intake.	Precedent Retching or Vomiting.	Blood Transfusion.	Diagnosis Made at	Gastro-Œsophageal Lesions.			
						Number.	Site.	Dimensions.	Depth.
Mallory and Weiss, 1929.	M. : 35	Heavy.	Yes.	No.[1]	Autopsy.	5	G.O.J.[2]	10–25 mm. × 1–2 mm.	Submucosa.
	M. : 31	Heavy.	Yes.	No.[1]	Autopsy.	3	Cardia.	To 15 mm. × 3 mm.	——
	M. : 56	Heavy.	Doubtful.	No.[1]	Autopsy.	2	Cardia.	15 mm. × 3 mm.	——
	M. : 61	Heavy.	Yes.	No.	Autopsy.	Several.	G.O.J.	10 mm. × 3 mm.	Muscularis.
Weiss and Mallory, 1932.	M. : 30	Heavy.	Doubtful.	No.[1]	Autopsy.	1	G.O.J.	2·5 cm. × 8 mm.	Muscularis.
	M. : 44	Periodic.	Yes.	No.	Autopsy.	1	G.O.J.	3·5 cm.	Perforated.
Decker, Zamcheck and Mallory, 1953.[3]	M. : 52	No.	——	?[1]	Autopsy.	1	Gastric.	——	——
	M. : 50	Heavy.	——	Yes.	Autopsy.	1	G.O.J.	——	——
	M. : 80	No.	No.	Yes.	Autopsy.	1	G.O.J.	0·5 cm.	——
	M. : 83	No.	No.	Yes.	Autopsy.	2	G.O.J.	1 cm.	——
	M. : 68	No.	Doubtful.	Yes.	Autopsy.	3	G.O.J.	1–1·5 cm. × 0·2 cm.	0·5–1 mm.
	M. : 65	?	——	?	Autopsy.	2	Gastric.	——	——
	M. : 50	Minimal.	No.	Yes.	Autopsy.	Several.	1 œsophagus and cardia.	2 cm. × 1·5 cm.	——
	M. : 75	Light — fairly heavy.	Yes.	Yes.	Autopsy.	3	Cardia.	1 cm.	——
	M. : 69	?	——	?[1]	Autopsy.	1	G.O.J.	——	——
	F. : 44	Alcoholic cirrhosis.	——	?[1]	Autopsy.	1	G.O.J.	1·5 cm. × 0·2 cm.	——
	M. : 57	Heavy — alcoholic cirrhosis.	Yes.	No.	Autopsy.	1	G.O.J.	3 cm.	——
Whiting and Barron, 1955.	F. : 79	No.	Yes.	Yes.	Operation.	1	Cardia.	5 mm.	——
Hardy, 1956.	M. : 21	None, except before vomiting.	Yes.	No.	Gastroscopy.	1	Cardia.	2 cm.	——
Small and Ellis, 1958.	M. : 44	Minimal.	Yes.	Yes.	Operation.	1	Lower part of œsophagus.	3 cm.	3 mm.
McPhedran, 1958.	M. : 40	Heavy.	Yes.	Yes.	Operation.	1	Lower part of œsophagus.	——	Submucosa.
Shuttleworth and Hutt, 1958.	F. : 53	?	No.	Yes.	Operation.	Multiple.	Lesser curve, from cardia, halfway to pylorus.	7–10 mm. × 1 mm.	Muscularis.
Mishkel and Jeremy, 1960.	M. : 68	? Moderate.	Yes.	Yes.	Autopsy.	2	Cardia.	2–2·5 cm. × 0·6–0·9 cm.	Muscularis.

[1] Required blood transfusion (B.T.) on data published.

[2] G.O.J. = gastro-œsophageal junction.

[3] In discussion (p. 961), however, it is stated that in " 5 cases in which the histories were sufficiently detailed vomiting preceded bleeding by an appreciable interval ".

A history of precedent severe retching and/or vomiting before the onset of bleeding is an important point in the diagnosis of this condition. In some cases it was difficult to assess whether there was an interval between the onset of vomiting and the bleeding; but in 14 of the cases this was well established, and in at least a further two cases there was a doubtful latent period. In the case reported by Hardy (1956) there was an interval of six and a half hours between the several attacks of vomiting and the subsequent hæmatemesis. In our case noisy eructations were a feature, and two large vomiting attacks preceded the hæmatemesis by approximately nine hours. Of special significance was the nurse's observation that only the last ounce of the patient's vomitus was blood-stained.

A total of 18 patients died, the vast majority of these as a result of exsanguination. In all, 11 patients were given transfusions. The first six patients were not given transfusions; but at least five of them would have received transfusions on present-day standards of treatment. The failure to give them transfusions was undoubtedly due to blood transfusions not being readily available during that period.

The amount of blood lost, by hæmatemesis and/or by melæna, in the majority of cases was large, amounting to many litres, for as much as 2 to 6·5 litres of blood were required to replace blood loss (Whiting and Barron, 1955). Although in many cases the bleeding took the form of the loss of bright red blood per rectum, in others the blood vomited was brown in colour, with episodes of vomiting fresh blood. However, in two cases there was no hæmatemesis or melæna. In Case 4 of Mallory and Weiss (1929), although the typical lesions were found at autopsy, there was no history of hæmatemesis or melæna, and the stomach and intestines at autopsy were free of blood. Again, in Case 2 of Weiss and Mallory (1932), there was no history of bleeding in the final illness; but at autopsy the fissured ulcer in the œsophagus had ruptured into the mediastinum with hæmorrhage into the right pleural cavity.

The lesions were remarkably uniform in size, shape and situation. Almost all were situated at the gastro-œsophageal junction, the majority being either distal to it or actually saddling the junction. However, in two cases the lesion was situated solely in the lowermost portion of the œsophagus (Small and Ellis, 1958; McPhedran, 1958). The one exception to this characteristic site was the case of Shuttleworth and Hutt (1958), in which multiple lacerations were present on the lesser curvature stretching from the cardia half-way to the pylorus.

The typical laceration is described as being in the long axis of the stomach and/or œsophagus, ranging from 1·0 to 3·5 cm. in length and 1 to 8 mm. in width, and extending down to the submucosa or the muscular coat. The second case of Weiss and Mallory (1932), in which the fissured ulcer saddling the gastro-œsophageal junction had ruptured through the œsophageal wall, demonstrated the close relationship between this syndrome and spontaneous rupture of the œsophagus.

The laceration may be solitary or multiple, as many as five lacerations being present in the first case recorded by Mallory and Weiss (1929). Multiple lacerations have also been recorded by Decker, Zamcheck and Mallory (1953) and by Shuttleworth and Hutt (1958) and in the present case.

In four cases the bleeding was described as coming from a ruptured artery in the laceration (Weiss and Mallory, 1932, Case 1; Whiting and Barron, 1955; McPhedran, 1958; present case, 1960), in one case from a ruptured arteriole (Mallory and Weiss, 1929, Case 3), and in another from a ruptured vein (Small and Ellis, 1958). In our case, surrounding one of the lacerations, there was extensive bleeding into the stomach wall, which could be seen both from the mucosal and serous surfaces. A similar state of affairs was found in Case 2 of Mallory and Weiss (1929), in which there was submucous hæmorrhage in the stomach and œsophagus. In a microscopic examination of the stomach mucosa between the lacerations, Shuttleworth and Hutt (1958) found that there was an extravasation of blood from the vascular plexus between the mucosa and the muscularis mucosæ, which subsequently led to the necrosis of the overlying mucosa. They held that this extravasation of blood was of importance in the pathogenesis of the fissure ulcers.

In the majority of cases, the histological picture was suggestive of an acute process, for the floor of the laceration was composed of an exudate of fibrin, polymorphs and red cells without any fibroblastic proliferation and new capillary formation. However, in Case 4 of Mallory and Weiss (1929), in Case 2 of Weiss and Mallory (1932) and in the case of Shuttleworth and Hutt (1958), early granulation tissue was in evidence. This histological picture is in accordance with the acuteness of the clinical picture, for the duration of the illness in the vast majority of cases, whether diagnosed at autopsy or at operation, was less than 10 days, and in more than half the duration was five days or less.

The association of chronic gastritis with the Mallory-Weiss syndrome has been stressed by Decker, Zamcheck and Mallory (1953), there being slight to severe gastritis in eight of their 11 cases. Histological proof of this was found in six of the eight cases in which specimens of gastric mucosa were available for microscopic study. They considered that the atrophic mucosa might be less resistant than normal mucosa to the traumatic effects of vomiting. However, as five of the six patients were aged over 50 years, they found that chronic gastritis was a difficult factor to evaluate because of the frequency with which atrophic gastritis is present in elderly people.

In microscopic examination of random sections of the stomach in the present case, one area showed a normal histological appearance and another area showed mild mononuclear infiltration.

The first essential in the management of these patients is the adequate replacement of the blood lost, both before and during the operation. In the case described by Whiting and Barron (1955) a total of 14 pints of blood were transfused, and in that of Shuttleworth and Hutt (1958) a total of 12 pints.

The second important point is to have a high index of suspicion of this condition in any case of hæmatemesis, especially in a middle-aged man who gives a recent history of vomiting and retching, and if the terminal portion of the vomitus is seen to be brightly blood-stained.

The value of endoscopy has been shown in one case reported by Hardy (1956), in which gastroscopy was performed whilst bleeding was still occurring and showed a two-centimetre oblique split in the mucosa of the posterior wall of the stomach about 3 cm. below the gastro-œsophageal junction, there being bright red blood oozing from the laceration. A barium-meal X-ray examination on the next day failed to show the lesion, as would be anticipated in view of the size, depth and situation of such a lesion. The rapid rate of healing of these lacerations can be realized, for a repeat gastroscopy seven days later showed no evidence of the laceration.

The Blakemore tube was unsuccessfully used in one case (McPhedran, 1958), and the author stated that although the tube would stop bleeding from an œsophageal varix, the 30 mm. of mercury pressure in the œsophageal balloon would be inadequate to control arterial bleeding. In this particular case a small vigorously bleeding artery was seen at laparotomy.

Laparotomy was performed in Case 2 of Weiss and Mallory (1932), and no lesion was seen. The abdomen was closed, and the patient subsequently died. At autopsy a saddle laceration of the gastro-œsophageal junction was seen. In the case described by Shuttleworth and Hutt (1958), a high Billroth I gastrectomy did not stop the bleeding, and the following day further resection of stomach and gastro-œsophageal junction was performed and five fissure-shaped ulcers were found in the remaining cardia and in the lower part of the œsophagus. These two cases teach a lesson—namely, that in all patients with hæmatemesis coming to laparotomy in whom the cause of bleeding is not evident, the cardio-œsophageal region should be examined visually, as these lesions cannot be palpated.

In three cases, the laceration was successfully sutured and the bleeding vessels were ligated. In all these cases there was only one laceration. In two cases the laceration was situated in the lower part of the œsophagus (Small and Ellis, 1958; McPhedran, 1958), and in the other case in the cardia (Whiting and Barron, 1955). Unfortunately any form of surgery was impracticable in our present case, because of the patient's poor condition following the myocardial infarction.

Summary.

A case of fatal myocardial infarction complicated by severe hæmatemesis is presented, together with the autopsy findings. The cause of the bleeding was found to be two fissure lacerations of the cardiac portion of the stomach. This case closely conforms to the syndrome described by Mallory and Weiss (1929). The literature concerning hæmatemesis from lacerations of the gastro-œsophageal region is reviewed.

Acknowledgements.

We wish to thank Dr. K. B. Noad for allowing us to publish this case, Dr. E. Hirst for his help with the autopsy report, and Miss J. Ashby, Librarian, for her assistance.

References.

DECKER, J. P., ZAMCHECK, N., and MALLORY, G. K. (1953), "Hæmorrhage from Gastroesophageal Lacerations at the Cardiac Orifice of the Stomach", *New Engl. J. Med.*, 249: 957.

HARDY, J. T. (1956), "Mallory-Weiss Syndrome. Report of Case Diagnosed by Gastroscopy", *Gastroenterology*, 30: 681.

MALLORY, G. K., and WEISS, S. (1929), "Hemorrhages from Lacerations of the Cardiac Orifice of the Stomach due to Vomiting", *Amer. J. med. Sci.*, 178: 506.

MCPHEDRAN, N. T. (1958), "Massive Upper Gastrointestinal Bleeding from Spontaneous Laceration of the Lower Œsophagus (Mallory-Weiss Syndrome)", *Canad. J. Surg.*, 2: 103.

SHUTTLEWORTH, K. E. D., and HUTT, M. S. R. (1958), "Mallory-Weiss Syndrome. A Case with Recovery after Total Gastrectomy", *Brit. J. Surg.*, 46: 1.

SMALL, A. B., and ELLIS, P. R. (1958), "Laceration of the Distal Esophagus due to Vomiting (The Mallory-Weiss Syndrome). Report of a Case with Massive Hemorrhage and Recovery after Repair of the Laceration", *New Engl. J. Med.*, 258: 285.

WEISS, S., and MALLORY, G. K. (1932), "Lesions of the Cardiac Orifice of the Stomach Produced by Vomiting", *J. Amer. med. Ass.*, 98: 1353.

WHITING, E. G., and BARRON, G. (1955), "Massive Hæmorrhage from a Laceration, Apparently Caused by Vomiting, in the Cardiac Region if the Stomach, with Recovery", *California Med.*, 82: 188.

Reviews.

Hypnosis in Anesthesiology. By M. J. Marmer, M.D., M.Sc.Med. (Anes.); 1959. Oxford: Blackwell Scientific Publications Limited. 9" × 6", pp. 166. Price: 54s.

OVER the past decade there has been a considerable increase in the use of hypnosis as an anæsthetic agent, particularly in dentistry and obstetrics, and to a lesser extent in general surgery. In these circumstances, it

would seem that the time is ripe for some authoritative guide on "Hypnosis in Anesthesiology"; this is the title of Dr. Milton Marmer's monograph.

The author starts with a readable account of the history of the use of hypnosis in anæsthesia, and there is special reference to the well-known work of Elliotson and Esdaile. There then follows an account of the induction of hypnosis. The approach is superficial. Various techniques for inducing hypnosis are clearly described; but no explanation is given of the underlying psychodynamics concerning what is happening to the patient during the process. No mention is made of such important matters as the patient's unconscious defences and how they may be circumvented. The actual words quoted to use with the patient often appear stilted. "All the tension has left your lower extremities and now you will be able to do the same with your upper extremities." Such expressions might well make the patient anxious, which is exactly what the author is trying to avoid. In fact, it would seem that the author's description of his relaxing technique would be very much improved by some explanation of the value of non-verbal communication with the patient.

In contrast with the rather inadequate description of the induction of hypnosis, the author's account of the use of hypnosis in anæsthesia is clear and well balanced. The psychological aspects of the induction of anæsthesia are discussed with sincerity and understandnig. The strength of the book lies in its moderation. Hypnosis is used to advantage in the pre-operative and post-operative phases. There is reduction in the amount of drugs used; but there is no attempt to replace pharmacological anæsthesia with hypnotic anæsthesia. However, the author concludes with a warning: "Do not force the situation—retreat. 1. If the patient becomes disturbed. 2. If anxiety develops. 3. If anything you cannot control arises." Surely these are situations which those who practise hypnosis should be trained to meet, and which should be discussed in a book on "Hypnosis in Anesthesiology".

Peripheral Facial Palsy: Pathology and Surgery. By K. Kettel, M.D.; 1959. Copenhagen: Ejnar Munksgaard, A.S. 10″ × 6¾″, pp. 341, with 127 illustrations. Price not stated.

THIS is a comprehensive coverage of the subject from the surgeon's viewpoint. In the foreword, Terence Cawthorne of London (himself a world recognized authority on surgery of the facial nerve) writes: "Dr. Kettel has made a great contribution to our knowledge of facial palsy and it is my belief that this mongraph will soon be found on the bookshelf of everyone who is interested in the subject." With this we readily concur.

The book is divided into three parts—the first "General Considerations", the second "Atraumatic Facial Palsies" and the third "Traumatic Facial Palsies". In the first part, the subject matter is taken step by step through the anatomical and physiological aspects. Degeneration and regeneration of nerves, general symptomatology and diagnosis, and operative methods available come into this part. In the second part, the author discusses the theories of causation of Bell's palsy in great detail, presenting a most enlightening treatise. Facial palsy associated with middle ear infections; herpes zoster oticus; Melkersson's syndrome and tumours come into this section. Finally, in the third section, all aspects of facial nerve palsy due to trauma are systematically discussed in detail.

The book is well set out, making easy reading, and fully illustrated. Perhaps a criticism could lie in the number of pictures of patients showing their "before and after" appearances; we might perhaps comment that the "sameness" of these photographs proves a little tiresome—at the same time, however, pointing out that this cannot and does not detract from the excellence of the text. The other illustrations are good, and there are some really excellent colour plates of operative fields. Then, in conclusion, we find a most comprehensive list of references, numbering approximately five hundred. This indicates the detailed study of the subject with which the author has made his approach to his task.

Pediatric Pathology. By Daniel Stowens, M.D.; 1959. Baltimore: The Williams & Wilkins Company. Sydney: Angus & Robertson, Limited. 10¼″ × 7¼″, pp. 684, with 374 illustrations. Price: £11.

THIS is the first comprehensive textbook of pædiatric pathology written in the English language. The reasons for the delay in the preparation of such a much-needed book are apparent. The field is a large one, which has undergone vast expansion in the past few years, and which is continuing to expand rapidly. As so many of the conditions seen by the pædiatric pathologist are malformations, such a book must also contain a considerable volume of embryology.

Stowens has made a valiant attempt to produce a volume which is up to date and includes all relevant embryological data. It is a volume that will be of great use to the pædiatric pathologist, and to the general pathologist who is faced on occasions with problems of pædiatric nature. As it is based on the immense store of material in the Armed Forces Institute of Pathology, it is obviously a comprehensive book. The appendix is an analysis of the first 10,000 cases entered in the files of the American Registry of Pediatric Pathology in the above-mentioned Institute, and is a very valuable addition to the book, although the selection is not that seen by the average pædiatric pathologist.

The author attempts to cover the entire subject, and much condensation has been necessary. Some portions of the book suffer badly on this score. It is difficult to imagine that the chapter on congenital heart disease will be of use to any pathologist. The references are an excellent feature of the book and well selected, although an occasional wrong reference was noted—for example, figure 219 (page 368), in which reference is made to the author's own work on melanotic adamantinoma.

The format of the book is pleasing and the style of the author attractive. The illustrations are mainly photomicrographs and their reproduction is excellent. The pathological aspects are integrated with a certain amount of clinical material, which adds to the value of the book.

Any pathologist experienced in the field of pædiatrics will find statements and hypotheses with which he would quarrel. For example, we would disagree with the type of cystic kidney which the author states is found in association with cystic disease of the liver and pancreas. Several unorthodox and unlikely theories proposed by the author could be quoted, but they do not detract from the essential value of this fine volume.

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"Pediatric Clinics of North America: Symposium on Recent Clinical Advances", edited by Vincent C. Kelley, M.D.; Volume 7, No. 2, 1960. Philadelphia and London: W. B. Saunders Company. Melbourne: Ramsay's Medical Books. 9″ × 5¾″, pp. 235 with illustrations. Price: £6 15s. (4 issues).

"The Demand for Medical Care: A Study of the Case-Load in the Barrow and Furness Group of Hospitals", by Gordon Forsyth, B.A. (Econ.) and Robert F. L. Logan, M.D., M.R.C.P.; 1960. London, New York and Toronto: Oxford University Press. 8½″ × 5½″, pp. 156. Price: 14s.

"Pathogenesis and Treatment of Occlusive Arterial Disease: The Proceedings of a Conference held in London at the Royal College of Physicians of London 13th-14th November 1959", edited by Lawson McDonald; 1960. London: Pitman Medical Publishing Co. Ltd. 9″ × 6″, pp. 252. Price: 39s. 6d. (English).

"The Discovery of Reflexes", by E. G. T. Liddell, D.M., F.R.S.; 1960. Oxford: The Clarendon Press. 9¼″ × 6″, pp. 184. Price: 54s. 6d.

"Practical Dietetics", edited by William A. R. Thomson, M.D.; 1960. London: The Practitioner. 9¼″ × 6″, pp. 64. Price: 4s. 6d. (English).

"Methods of Biochemical Analysis", edited by David Glick; Volume 8, 1960. New York: Interscience Publishers Inc., and London: Interscience Publishers Ltd. 9″ × 6″, pp. 412. Price: $10.00.

"Viral Infections of Infancy and Childhood: A Symposium of the Section on Microbiology, The New York Academy of Medicine", edited by Harry M. Rose, M.D.; 1960. New York: Paul B. Hoeber, Inc. Medical Division of Harper & Brothers. 9″ × 5¾″, pp. 256 with illustrations. Price: $8.00.

"The Year Book of Dermatology", edited by Rudolf L. Baer, M.D., and Victor H. Witten, M.D.; 1960. Chicago: The Year Book Publishers. 7¼″ × 5″, pp. 480 with illustrations. Price: £4 19s.

The Medical Journal of Australia

SATURDAY, AUGUST 27, 1960.

THE GENETIC BASIS OF THALASSÆMIA.

ON p. 328 of this issue, J. G. Wilson discusses the increasing frequency with which thalassæmia is being encountered in Adelaide, as no doubt elsewhere in Australia, as a result of the increasing number of people of Mediterranean stock in our population. A number of curious facts about this disease have long intrigued medical scientists, and though it is now recognized as definitely an hereditary condition, there is still much speculation as to the exact mechanism of its inheritance.

Many of us can remember the days when hæmatology appeared to be a relatively straightforward subject, mainly concerned with the diagnosis of the various kinds of anæmias and blood dyscrasias. Hæmatologists have long since then moved into an atmosphere which is so rarified that only the boldest essay to follow them, and in which nothing is as simple as it at first appears. Nevertheless, for those who try to keep abreast of the science of medicine, to attempt to follow them in a new synthesis, such as that put forward by V. M. Ingram and A. O. W. Stretton[1] in seeking to explain the genetic basis of the thalassæmia diseases, is a stimulating and interesting exercise, involving as it does various facets of advanced research. Ingram and Stretton do not claim originality for their hypothesis, as it takes in various ideas already widely discussed by those who move in the esoteric realms where the boundaries between hæmatology, biochemistry and genetics disappear; however, they accept complete responsibility for the final synthesis as they propound it.

Ingram and Stretton begin by pointing out that research in recent years has made it clear that the abnormal hæmoglobins, of which quite a number are now recognized, are variants of the normal hæmoglobin *A*; some have been shown to differ from hæmoglobin *A* in the substitution of a single amino-acid residue by a new one, and their inheritance is controlled by a single gene. One of the chief methods of distinguishing these abnormal hæmoglobins is by their electrophoretic behaviour. Thalassæmia on the other hand, though inherited in a similar manner, presents a more complicated and confusing picture. In thalassæmia there is no recognizably abnormal form of hæmoglobin present, but anæmia appears to be due to an interference with the rate of production of hæmoglobin *A*. Ingram and Stretton seek to explain the occurrence of thalassæmia and some of its interactions with the other abnormal hæmoglobinopathies (in which chemically abnormal hæmoglobins are recognized) by suggesting that thalassæmia is caused by a mutation producing a hæmoglobin with a "hidden" amino-acid substitution, such that the hæmoglobin produced is electrophoretically normal.

The basic facts about thalassæmia are well known. It is an hereditary disease, which occurs commonly in Mediterranean peoples, and is found in two forms—the major form, which is characterized by marked hypochromic anæmia with abnormal red-cell morphology, and the minor form, in which the degree of anæmia is slight or non-existent, and which is diagnosed chiefly by minor hæmatological abnormalities. Thalassæmia major is the homozygous form of the condition, and it is commonly found that the parents of a patient with the major form can both be shown to be subjects of thalassæmia minor, though often in an asymptomatic form. It has further been shown that parents of a child with thalassæmia major have a slightly increased proportion of hæmoglobin A_2, a normal constituent of hæmoglobin which usually makes up 2·5% of the total hæmoglobin. However, there are exceptions to this, and Ingram and Stretton state that these, together with the great variability of the disease in general, suggest that there is not one kind of thalassæmia (major or minor) but several. Another feature of thalassæmia major is that fœtal hæmoglobin, which is normally almost entirely replaced by hæmoglobin *A* in the first few months of life, may contribute as much as 97% of the total hæmoglobin in patients with this condition, instead of the normal figure of about 1%. This is assumed to be a compensation for the marked decrease in the amount of normal adult hæmoglobin synthesized in such patients. Further light on the nature of the disease is obtained from a study of cases in which thalassæmia minor is combined in the one patient with the inheritance of the trait for one of the abnormal hæmoglobins. For example, in individuals who are heterozygous for, say, hæmoglobin *S*, both normal hæmoglobin *A* and hæmoglobin *S* are present in the blood, usually with a preponderance of hæmoglobin *A*. This is taken to indicate that the rate of synthesis of hæmoglobin *A* is greater than that of hæmoglobin *S*, on the assumption that hæmoglobin synthesis is more or less complete before the red cells enter the circulation. However, if a person is heterozygous for both hæmoglobin *S* and the thalassæmia gene, the proportion of hæmoglobin *S* present is about double that found in the first instance, and the remainder is largely fœtal hæmoglobin. This appears to indicate a specific inhibition of hæmoglobin *A* synthesis with little or no interference in the production of the abnormal hæmoglobin, and to rule out the hypothesis that thalassæmia is caused by some enzymatic defect which interferes with one of the steps in the synthesis of the hæm element. Study of such cases has also suggested that the genes which determine the type of hæmoglobin produced also control the rate at which it is produced. Ingram and Stretton illustrate this by stating: "thus, a gene controlling the production of a globin with a certain amino-acid sequence will effect synthesis of protein at a certain rate, while a gene associated with a globin having

[1] *Nature* (*London*), 1959, 184: 1903 (December 19).

the same sequence except for one amino-acid may have a different rate of synthesis." They then state that it has been postulated, as an explanation of the varying ratios of hæmoglobin *A* to hæmoglobin *S* in heterozygous subjects, that normal hæmoglobin is controlled by a series of at least three alleles, which produce hæmoglobin at different rates. They propose to extend this hypothesis to include thalassæmia, postulating that the apparently normal hæmoglobin found in thalassæmia homozygotes has a different amino-acid sequence from hæmoglobin *A*, and that this new sequence determines a characteristically low rate of synthesis.

From here Ingram and Stretton elaborate their hypothesis, pointing out that it is now believed that hæmoglobin has four polypeptide chains of two different kinds (α and β chains), and that abnormalities may arise in either the α chains or the β chains, giving rise to a great variety of possible combinations of normal and abnormal chains. It is also believed that each hæmoglobin peptide chain is controlled by a specific gene, the production of hæmoglobin being therefore controlled by two sets of genes. A surprising amount is already known about this; for example, it is believed that in the sickle-cell trait, the abnormality is due to a mutation in the β chain, while another abnormal hæmoglobin, hæmoglobin *I*, is caused by a mutation in the α chain. Ingram and Stretton put forward the suggestion that thalassæmia may be due to a mutation in either the α or β chain, which differs from the mutations which lead to the production of abnormal hæmoglobins such as *S* or *C* only in that the consequent amino-acid alteration does not change the electrophoretic behaviour of the hæmoglobin produced, or is such that the chain involved is never made at all, and therefore cannot be observed. It is clear that several, possibly many, different mutations might thus produce results distinguishable only by the severity of the resulting defect.

Ingram and Stretton then proceed to examine various relevant family studies which have been published, in the light of their hypothesis. They also mention an alternative hypothesis, the "tap" hypothesis, according to which the defect in the thalassæmia gene acts not on the type of hæmoglobin produced, but merely on the rate of production of one of the normal polypeptide chains. They state that the genetic analysis presented by them is valid in terms of both hypotheses, but that they have chosen to develop the substitution hypothesis in detail because it is based on fewer assumptions than the "tap" hypothesis. They point out that their hypothesis is open to experimental test, and express the hope that, even if it is proved wrong, it will serve to stimulate interest and to direct experiment in this field. It is perhaps only fair to state Ingram and Stretton's summary in their own words:

> We propose that thalassæmia is a mutation of either the α or β hæmoglobin gene, of the same kind as that observed in the abnormal adult hæmoglobins, but without effect on the electrophoretic behaviour of the protein. Such mutations would be expected to reduce, sometimes to nothing, the rate of the production of that hæmoglobin.

Before leaving the subject of hæmoglobin we should like to draw attention to another example of virtuosity in the more rarified planes of research. It is now possible, by refinements of techniques beyond the reach of the imagination of ordinary mortals, to construct with the help of X-ray crystallography contour models to show the actual shape of complex molecules. In an exercise of this sort and with the help of their biochemical knowledge, M. F. Perutz and his colleagues[2] at the Cavendish Laboratory, Cambridge, have constructed a scale model of the hæmoglobin molecule, showing the four polypeptide chains elaborately coiled around their respective hæm groups in a fixed pattern. It is difficult to convey an idea of the complexity of these models without showing the actual picture. It is perhaps a test of our capacity for scientific wonderment whether we regard such achievements, which have cost a group of first-class minds many, many hours of painstaking research, as merely another clever trick or as another step towards the elucidation of the chemical nature of life itself.

Current Comment.

THE TRANSMISSION OF LEPROSY.

It has long been recognized that leprosy is not easily transmitted, and generations of students have been brought up to believe that the infection is acquired only after "prolonged and intimate contact" with the disease. This second belief is challenged, and the factors which influence the transmission of leprosy are reviewed, in an article by J. A. Kinnear Brown, published in the *Transactions of the Royal Society of Tropical Medicine and Hygiene* and reprinted in the *International Journal of Leprosy*.[1] This review is based on seven years' work on leprosy in East Africa, including a large number of surveys carried out in Uganda and parts of Kenya. Kinnear Brown begins by listing what he terms "some of the more traditional views" about the incidence and transmission of leprosy, and then comments on them one by one. He has no difficulty in showing that climate, density of population and overcrowding have little effect on the prevalence of the disease. Commenting on the belief that leprosy is commoner among males, he states that in Uganda this appeared to be so only because men come to hospital more readily than females; surveys showed a similar incidence in both sexes. However, it is agreed that lepromatous leprosy is commoner among males, and the tuberculoid type among females, and Kinnear Brown suggests that the sex incidence in any one area may reflect the relative incidence of the two main types of the disease. The age distribution of the disease in Uganda shows two peaks, one between the ages of 10 and 15 years, the other in the 25 to 30 years age group. A similar fall in incidence in the 15 to 19 years age group has been recorded in previous surveys in Uganda and in Dutch New Guinea, and Kinnear Brown attributes the double peak to the existence of two periods of infection, one in childhood, involving principally the children of infected parents, and one in early adult life, when many young adults leave their homes and come in contact with infection for the first time. However, quite a number of patients have shown no symptoms till middle life, and some of Kinnear Brown's figures suggest a picture analogous to that of tuberculosis in the advanced countries, with a shift in incidence to the later decades. Kinnear Brown concludes that the age of infection depends very much on opportunity, and that the incidence in childhood should be considered in this light.

An interesting point brought out by the surveys is that in a number of instances the rate in isolated ethnic groups was several times that of the surrounding tribes of different stock. A curious parallel is provided by the example of a small group of colonists of German origin who settled in Venezuela about 100 years ago, and have

[2] *Nature (London)*, 1960, 185: 416 (February 13).
[1] *Int. J. Leprosy*, 1959, 27: 250 (July-September).

maintained their isolation and their culture by keeping their stock pure; in a community of a little over one thousand souls there were said, in 1952, to be over 100 lepers. It seems likely that such an example could be explained by local and historical factors, but Kinnear Brown uses these instances in support of a theory of genetic susceptibility, some of the arguments for which are not very easy to follow.

However, he seems to be on firmer ground when he attacks the conventional notion that the tuberculoid (non-lepromatous) type of Hansen's disease is non-infectious. In Uganda, though the population is fairly large it is predominantly rural, and is unusually dispersed in that the peasant farmers live each on his own plot of land rather than in villages. This provides an exceptional setting for the study of the epidemiology of the disease, and Kinnear Brown shows that it is difficult to account for the distribution of cases unless one assumes that non-lepromatous patients may at times become infectious. There is no doubt that the lepromatous patient, with his skin and mucous membranes teeming with bacilli, is the more grossly infective, but Kinnear Brown produces strong evidence to suggest that tuberculoid patients may also at times transmit the infection, and in some circumstances may even be the most important sources of fresh infections. He regards the relaxation of segregation in non-lepromatous cases as a retrograde step, and suggests that only the introduction of modern chemotherapy has neutralized the dangers of this change in policy.

There are many anomalies about the spread of leprosy. Kinnear Brown comments: "The rate of conjugal infection ought to be high, it is surprisingly low. All the children born to patients should contract the disease; many of them do not. There should be a high rate of infection among those who are in daily contact; it is far lower than one would expect." Because of these and other facts Kinnear Brown believes that infection occurs because of susceptibility, a composite state which may vary during the life of the individual and is governed by several factors, some or all of which are genetically determined. Moreover, for the susceptible individual exposure does not have to be prolonged or intimate. Contact there must be, but the only other necessities may be an individual in a susceptible phase and a patient in an actively infective condition. As Kinnear Brown remarks, the phrase "prolonged intimate contact" merely covers up something which we do not understand about the transmission of the disease. "Using it does not solve the difficulty. Some form of intimate contact is reasonable, but how intimate and why prolonged?" This controversy is reminiscent of the history of opinions on the epidemiology of tuberculosis. At one time great importance was attached to familial susceptibility; at a later stage it was taught that this was merely the manifestation of familial exposure to infection; more recently, however, there is a tendency to reconsider the role of natural susceptibility in the spread of infection. There is still undoubtedly much to be learned about the epidemiology of leprosy, but now that the disease is coming increasingly under control, it is not unlikely that some of its enigmas will never be solved, but will die with the disease which created them.

DOCTOR JENNER OF BERKELEY.

BEFORE the middle of the eighteenth century, doctors gave serious attention to the prolific lucubrations of ancient writers on the mysterious workings of the human body and the bizarre measures confidently recommended for the curing of all disease. Then came the exercise of independent thought, a firmer reliance on experimental methods and the accurate observation of facts as they presented themselves in the investigation, so that in the second half of the century a few men imbued with the scientific spirit succeeded in opening up the way for progress in the unexplored realm of preventive medicine. Outstanding among these benefactors of the human race was Dr. Edward Jenner, the country general practitioner of Berkeley in Gloucestershire, whose special contribution was the discovery of a simple method for the prevention of the dreaded smallpox, which is said to have accounted for some 40,000 deaths annually in England alone. The latest biography of this great man[1] is written by Dorothy Fisk, a writer of popular books with a scientific flavour, who has undertaken a great deal of careful research on the subject and tells her story with poetical insight, a fluent style and a wealth of reliable material attractively presented to satisfy the general reader.

Edward Jenner was always a great student and admirer of the works of nature, a lover of beauty, well trained and instructed in the art and science of medicine and loyal and affectionate in his long friendships, and he never failed in his devotion to the interests and welfare of his own family circle. One of the greatest pleasures and interests of his life was the close association and deep friendship with the famous surgeon, John Hunter, who remained on terms of intimacy with his favourite pupil. He had the same passion for delving into the dark mysteries of natural history and gave every encouragement to the country practitioner to pursue his studies of the local fauna and flora, while continually urging him to write down and publish the results of his original research.

In 1788, Jenner's paper on the life history of the cuckoo earned for him the coveted honour of election to Fellowship of the Royal Society of London, and in 1798 was published his small book "An Inquiry into the Causes and Effects of the Variolæ Vaccinæ", which gave clear directions for a safe, reliable and effective method for protecting the individual against smallpox. While foreign potentates and political leaders in other parts of the world were anxious to bestow the highest honours upon this benefactor of the human race, he had to wait a long time for his fame to be acknowledged in England. In fact, his own country persistently maintained a conservative attitude to the discovery, contested his right to be relieved of financial anxiety and argued about his claim to priority as the true prophet of vaccination. Consequently, it was many years before the dangerous practice of variolation was entirely abandoned.

It is interestingly recalled that the three friends, John Hunter, Edward Jenner and Caleb Hillier Parry of Bath, were true moderns in the investigation of disease. Parry was to give the first description of hyperthyroidism as a definite clinical entity, and Jenner had made his own observations on the signs and symptoms of coronary sclerosis after his dissections had shown evidence of hardening in the arteries of the heart. In view of these findings Jenner had reluctantly avoided following his usual procedure of communicating with John Hunter to invite discussion on any new problem. Instead, he decided to refer his observations to Dr. Heberden of London, with these remarks: "Should it be admitted that this is the cause of the disease, I fear the medical world will seek in vain for a remedy, and I am fearful (if Mr. Hunter should admit this to be the cause of the disease) that it may deprive him of the hope of recovery."

Finally, it might be pointed out in the interests of historical accuracy that Sir Gilbert Blane was a distinguished naval physician and is unlikely to have held the position of President of the Royal College of Surgeons in London; and that the Mr. Stephens referred to as having received a grant of £5000 from the English Government was a Mrs. Joanna Stephens, whose magical formula for dissolving bladder stones not only hoaxed the legislature but had a supporter in William Cheselden, the leading surgeon of his day.

One topical comment seems relevant and justifiable. The threat of serious outbreaks of smallpox is still a reality among crowded populations in many underdeveloped countries, and this book may serve a useful purpose as a reminder that there is still a need for constant vigilance and that the Jennerian method of vaccination is not yet obsolete.

[1] "Doctor Jenner of Berkeley", by Dorothy Fisk; 1959. London, Melbourne, Toronto: William Heinemann Ltd. 8¾" × 5¾", pp. 296, with illustrations. Price: 31s.

Abstracts from Medical Literature.

OPHTHALMOLOGY.

Demecarium Bromide and Echothiophate Iodide in Chronic Glaucoma.

B. BECKER AND T. GAGE (*A.M.A. Arch. Ophthal.*, January, 1960) present their observations on the use of demecarium bromide and echothiophate iodide in a group of glaucoma patients not controlled by other forms of therapy. Demecarium bromide was used in 144 eyes of 76 patients and echothiophate iodide therapy was started in 155 eyes of 83 patients. Patients were treated with 0·25% solution instilled once daily and in those who failed to respond demecarium bromide was used in 1% solution. Demecarium bromide 0·25% solution improved facility of outflow and lowered intraocular pressure in about 50% of eyes not previously controlled. When 1% solution was used for those which failed to respond to 0·25%, the percentage improved to 54. Echothiophate 0·25% also controlled intraocular pressure in 54% of another group. Ocular side effects were ciliary and conjunctival injection, blurring of vision, ocular and periorbital pain and headache. These symptoms rarely persisted after one week. Bilateral retinal detachment occurred in two patients. Systemic side effects were nausea, vomiting, diarrhœa, bradycardia, salivation and sweating. Failure to recognize the systemic effects of these drugs has resulted in extensive unnecessary medical and X-ray investigations.

The Applanation Tonometer.

J. AMDUR (*A.M.A. Arch. Ophthal.*, January, 1960) describes the use of the applanation tonometer. As the use of the applanation tonometer displaces only 0·45 cu. mm. of fluid there is negligible distension of the ocular coats, and therefore scleral rigidity is insignificant. A second advantage is that the intraocular tension can be measured with the patient in the sitting position without moving the patient from the slit lamp. The author gives a detailed step by step description of the use of the applanation tonometer. With fair patient cooperation the entire procedure can be accomplished in two to three minutes after the cornea has been anæsthetized.

Intraocular Nævoxantho-Endothelioma.

A. E. MAUMANEE AND D. W. LONGFELLOW (*Amer. J. Ophthal.*, January, 1960) report on two cases of juvenile xanthogranuloma. In both cases the iris lesion subsided under treatment. In one case the lesion did not appear until the age of three and a half years (in most other cases reported the lesion has been noted by six months). The diagnosis was made on the presence of repeated anterior chamber hæmorrhages and the lesion in the iris—a yellow-brownish or salmon coloured, diffuse, patch-like tumour of the iris. Biopsy of the iris showed cells resembling histiocytes. In the second case biopsy was not performed on the iris lesion, but the skin lesions were diagnosed as nævoxantho-endothelioma. In both cases X-ray therapy was used on the basis that the disease resembled the reticulo-endotheliosos, and it had been noted that isolated lesions of Christian-Schiller disease have regressed with X-ray therapy. In other cases reported, the eye had been lost from secondary glaucoma, and the authors considered that any treatment which might cause regression of the tumour was justified if secondary glaucoma was to be averted. The treatment was given in divided doses over five days. In addition, cortisone therapy was given locally and systemically. The authors state that the disease should be suspected when a child has a history of repeated spontaneous anterior chamber hæmorrhages, and diffuse thickening of the iris with yellow-brown or salmon-coloured patch-like lesions. The ciliary body may be involved. Keratic precipitates are present. Treatment should be instituted immediately, and consists of topical and systemic cortisone therapy. Secondary glaucoma should be controlled with "Diamox" and miotics, and if it cannot be controlled, X-ray therapy should be given. A single dose should not exceed 200*r* to the lens, and preferably should not be more than 150*r*. If any more radiation is required, an interval of two or three weeks should elapse.

Beta Radiation in Ophthalmology.

B. D. LEAHEY (*Amer. J. Ophthal.*, January, 1960) discusses the indications for beta radiation in ophthalmology. The author considers this therapy particularly useful for the removal of corneal blood vessels, rosacea keratitis, vernal conjunctivitis when steroid therapy has failed, and recurrent pterygium, in which condition the author regards beta radiation as a specific cure. This form of therapy is contraindicated for interstitial keratitis, corneal scars, granuloma, epithelial downgrowth in the anterior chamber, and epithelioma. The complications of radiation therapy are effects in the lens (radiation cataract), and on the conjunctiva, cornea and sclera, where it may cause transient or subacute irritation, telangiectases, keratinization of the conjunctival epithelium, chronic irritation and ulceration or late ulceration of the cornea, which may appear years after the completion of successful therapy.

Pathological Effects of an Anterior Chamber Acrylic Implant.

N. ASHTON AND D. P. CHOYCE (*Brit. J. Ophthal.*, October, 1959) present a report on the pathological examination of a human eye obtained six weeks after the insertion of an anterior chamber acrylic implant. There was a chronic inflammatory reaction at the limbus, and three healed perforating wounds passed obliquely through the periphery of the cornea. At the inner aspect of one of these wounds the cornea was denuded of Descemet's membrane. Schlemm's canal and the uveo-scleral network appeared healthy; the filtration angles were open and on both sides the root of the iris had been pushed backwards. The implant had considerably distorted the tissues of the corneo-iridic angles to form cups of compressed atrophic tissue extending outwards and backwards into the ciliary body. In this tissue there was a mild inflammatory reaction. On the temporal side a folded strip of Descemet's membrane lay buried in the tissues of the iris root. There was some degree of macular œdema. From these findings the authors conclude that damage to the endothelium may be not inconsiderable, and that if this is excessive, it may produce endothelial dystrophy. Surgical technique should therefore be gentle. To prevent possibility of contact of lens and endothelium patients should be warned not to press on the eye, say when washing the face. Stretching and atrophy of structures of the angle might be lessened if the thickness of the haptic portion of the implant could be reduced. The inflammatory reaction in the eye was surprisingly mild. However, glaucoma secondary to anterior uveitis could be a possible complication. There was no evidence of obstruction to aqueous outflow from blockage of the angle by the implant or by fibrosis of the uveo-scleral meshwork. The finding of macular œdema was unexpected.

Light Coagulation.

D. GUERRY AND H. WEISINGER (*Amer. J. Ophthal.*, January, 1960) describe their experiences with the use of light coagulation in 42 eyes. The technique was used with good result in retinal tears with or without retinal detachment. In addition macular holes and cysts were cured. The use of light coagulation in classical detachment surgery, either at the end of surgery or some days later, proved to be effective. Other conditions which responded favourably were retinal and choroidal tumours, neovascularization, central retinal schisis and solitary acute choroiditis. External diseases were also treated with good effect. The authors regard the technique as a valuable therapeutic technique.

Fistulizing Operations for Glaucoma.

I. H. LEOPOLD (*J. int. Coll. Surg.*, February, 1960) discusses the reasons for failure of fistulizing operations in glaucoma. Apart from improper choice of operation, these are complications that may occur with the performance of a properly selected procedure. Complications are divided into early and late. The early complications are hyphæma, shallow and flat anterior chamber, vitreous hæmorrhage, uveitis, cataract, retraction of iris pillar in iris inclusion and malignant glaucoma. Rare complications are uncontrolled tension, progressive field loss, cataract, hypotony, drawn-up pupil, corneal œdema, uveitis and intraocular infection. In spite of the possibility of complications, operation is indicated if the patient has a rising base pressure, evidence of field loss and decreased facility of outflow. Complications are likely to occur no matter which procedure is used.

Dacryocysto-Rhinostomy with Wire Fistulization.

I. A. ABRAHAMSON, JR, AND I. A. ABRAHAMSON, SR (*Amer. J. Ophthal.*, December, 1959) describe their technique of dacryocysto-rhinostomy with wire fistulization and the results in 30 patients.

An incision 1·5 cm. long is made through the skin 3 or 4 mm. on the nasal side of the inner canthus. Starting at the fold of the upper lid, the incision is carried 3 mm. below the orbital rim down to the nasal bone. The sac is freed from the lachrymal fossa. A window is made in the naso-lachrymal bone with a Stryker bone saw using a blade 10 mm. in diameter. A longitudinal incision is made in the nasal mucosa. A similar incision is made in the posterior or inner wall of the sac. A Veirs needle is passed through the lower punctum, opening in the sac, and the nose. The stylet is removed and a 20 gauge stainless steel wire is passed through the Veirs needle into the nose. The Veirs needle is removed and the wire united. The anterior and posterior edges of the incision in the sac are sutured to the corresponding edges of the nasal mucosa using 4/0 chromicized gut. Subcutaneous tissue and skin are then sutured. The steel wire is jiggled in a circular motion for one minute five times a day for 21 days. The naso-lachrymal passages are irrigated with penicillin after the wire is removed and again two weeks later.

OTO-RHINO-LARYNGOLOGY.

Acoustic Neuroma.

M. R. DIX AND C. S. HALLPIKE (*Laryngoscope* (*St Louis*), February, 1960) discuss what they consider to be the important factors in the diagnosis of acoustic neuromas. They divide their cases into those of true eighth nerve neurofibromas, which arise from the neurilemma in the porus acusticus, and those of other pontine angle tumours, which later involve the eighth nerve. They point out that in the former there is a definite otological period before extension of the tumour leads to other neurological findings. Cochlear function is discussed under the subheadings "Hearing Loss", "Loudness Recruitment" and "Bone Conduction", and vestibular function under the subheadings "Stance and Gait", "Spontaneous Nystagmus", "Positional Nystagmus" and "Caloric Tests". Finally, the authors try to answer some questions which are put to them from time to time regarding the clinical value of modern diagnostic tests.

Tracheostomy.

T. CAWTHORNE, A. B. HEWLETT AND D. RANGER (*Proc. roy. Soc. Med.*, June, 1959) present a review of the historical development of tracheostomy with an explanation of its present position. This procedure was first mentioned by Asclepiades in the first century B.C. The word "tracheotomy" was first used in 1649 by Thomas Fienus, and the term "tracheostomy" was suggested by Negus in 1938. The procedure was reserved for cases of obstruction at or above the level of the larynx until Wilson, in 1932, and Davidson, in 1936, suggested tracheostomy for the relief of respiratory insufficiency due to the bulbar form of poliomyelitis. About this time, this procedure was also suggested as an alternative to repeated bronchoscopy in acute laryngo-tracheo-bronchitis. In 1943, Galloway strongly advocated tracheostomy for respiratory emergencies regardless of the cause, and it was the poliomyelitis epidemic in Denmark in 1952 that finally convinced the medical world of the validity of this philosophy. In 1940, 98% of all tracheostomies were performed for relief of laryngeal obstruction, whereas in 1958 only 48% were performed for this reason. In other words, more than half of the tracheostomies now performed are for the prevention of or to overcome respiratory insufficiency from causes other than laryngeal obstruction. The indications for tracheostomy are summarized under the following headings: (i) to relieve obstructive laryngeal dyspnœa; (ii) to facilitate removal of secretions retained within the tracheo-bronchial tree; (iii) to discourage anything but air from entering the tracheo-bronchial tree; (iv) to assist respiration by reduction of the dead space; (v) to isolate the respiratory tract from the digestive tract; (vi) to permit positive-pressure respiration.

Treatment of Facial Palsy.

K. KETTEL (*A.M.A. Arch. Otolaryng.*, April, 1960) discusses a recent paper by Martin and Helsper in which these authors tried to present clinical evidence that, after surgical section and sacrifice of a considerable segment of the seventh cranial nerve (including a portion of its main trunk and the peripheral plexus), there could be spontaneous recovery of function in a fair percentage of cases without resort to nerve grafting or any other form of neurorrhaphy; they questioned the justification for any form of neurorrhaphy or extensive plastic repair for seventh cranial nerve paralysis due to operative defects until at least a year or more has passed. Martin and Helsper postulated that, after complete and permanent interruption of the motor pathway through the seventh cranial nerve, voluntary motor impulses by reeducation could find their way from the cortex through the fifth cranial nerve to the muscles involved. Kettel states that this proposition is in flagrant contradiction to physiological and neurological laws hitherto accepted, and points out that immeasurable harm will be done to patients suffering from atraumatic facial palsy if Martin and Helsper's advice is taken.

Chronic Otitis Media and Mastoiditis.

D. MYERS AND W. D. SCHLOSSER (*Laryngoscope* (*St Louis*), January, 1960) present a preliminary report of what they term the anterior-posterior technique for surgical treatment of chronic middle ear disease and mastoiditis. They point out that otologists have always found trouble in that the post-operative mastoid cavity tends to fill with granulation tissue and close over. They therefore suggest an operation in which first a routine post-auricular mastoid operation is done, going as far forward in the region of the additus as possible and exposing the epitympanic space with an ear speculum and operating microscope, the middle ear is explored and diseased tissue can be removed. They state that the technique can be varied to perform myringoplasty and tympanoplasty grafting and plastic procedures. They leave a piece of fine "Polythene" tube which leads through the posterior wound into the attic region. This is left in for some weeks, and antibiotic solution can be instilled through this tube. The authors state that their technique results in a dry ear with improved hearing.

Nasal Fractures.

A. HURST (*Laryngoscope* (*St Louis*), January, 1960) gives a résumé of 100 cases of nasal fracture treated at his hospital. He comments that the results are not good if one looks for lack of X-ray evidence of displacements and complete absence of cosmetic deformity after reduction. He thinks that the poor results are due to poor post-reduction splinting and internal packing. He classifies the fractures into simple (greenstick), linear without displacement, linear with either lateral displacement or depression and comminuted open fractures. He states that presence or absence of crepitus and the results of X-ray examination are two pieces of evidence which can be quite misleading. Comminuted open fractures gave the worst results. The author gives a fairly detailed method of treatment for all types of cases, and also makes special mention of the problem in children.

Agammaglobulinæmia.

P. A. WALLENBORN (*Laryngoscope* (*St Louis*), January, 1960) presents a detailed account of agammaglobulinæmia and reviews the literature. He reports a typical case of acquired agammaglobulinæmia in a young girl, and of congenital agammaglobulinæmia in a young boy. The author states that the otolaryngologist often has the first opportunity to make the diagnosis of agammaglobulinæmia, whether it is the congenital or the acquired type, and should strongly consider this diagnosis in any patient with recurring acute or chronic infections of the respiratory tract. He should also think of this diagnosis in any patient showing a striking deficiency or absence of tissue. Paper electrophoresis of the serum proteins, which is now generally available, will usually establish the diagnosis. Other presumptive clinical, laboratory and X-ray findings are reviewed.

Cholesteatoma Formation in the Middle Ear.

L. RUEDI (*Acta oto-laryng.* (*Stockh.*), May-August, 1959) reports on the experimental production of cholesteatoma in animals. He has produced typical aural cholesteatoma in guinea-pigs behind an intact tympanic membrane. This was in response to a mild foreign body reaction. The cholesteatoma develops as a result of proliferation of basal epidermal cells of the tympanic membrane growing into the granulation tissue of the cavum tympani. Breakthrough into the external auditory meatus is secondary. These experiments support Lange's views of the causes of human cholesteatoma with small upper marginal perforations. This postulates immigration of the epidermis in response to an inflammatory stimulus. A cholesteatoma thus formed behind an intact drum breaks into the external auditory meatus at a later stage, thus giving rise to a small marginal perforation.

British Medical Association.

SOUTH AUSTRALIAN BRANCH: ANNUAL MEETING.

THE annual meeting of the South Australian Branch of the British Medical Association was held on June 29, 1960, in the Memorial Hall, 80 Brougham Place, Adelaide, Dr. G. T. Gibson, the President, in the chair.

ANNUAL REPORT OF THE COUNCIL.

On the motion of Dr. R. M. Glynn, seconded by Dr. P. Verco, the annual report of the Council was received and adopted. The report is as follows.

The Council presents the following report of its activities during the past year.

At the annual general meeting of the Branch held on June 24, 1959, the following officers and members were elected for the ensuing year:

President: Dr. G. T. Gibson.

Vice-President: Dr. R. G. C. de Crespigny.

Honorary Treasurer: Dr. J. M. Dwyer.

Honorary Medical Secretary: Dr. Robert Hecker.

Ordinary Members of Council: Group A (metropolitan), Dr. E. P. Cherry, Dr. I. S. Magarey, Dr. H. R. Oaten. Group B (country), nil.

At the first Council meeting of the new year, held on July 2, 1959, Dr. W. J. Sleeman of Renmark was invited to fill the vacancy caused by the failure to elect until the date of the next annual meeting, *vide* Rule 43 (2).

The following sub-committees were also appointed:

Scientific: Dr. Bonnin and Dr. I. S. Magarey.

National Health Service: Dr. Mallen, Dr. Rieger, Dr. Heddle and Dr. E. P. Cherry.

Ethics: Dr. Rieger, Dr. Dwyer, Dr. Jungfer, Dr. Heddle, Dr. de Crespigny and Dr. Oaten.

Parliamentary Bills: Dr. Mallen and Dr. Rieger.

Library: Dr. Jungfer, Dr. Hecker, Dr. de Crespigny and the Honorary Treasurer (*ex officio*).

Salaries: Dr. Mallen, Dr. Rieger and Dr. Oaten.

Medico-Pharmaceutical Liaison Committee: Dr. Hecker, Dr. Rieger and Dr. N. Halloran.

Tuberculosis Standing Committee: Dr. R. C. Angove, Dr. J. L. Hayward, Dr. K. S. Hetzel, Dr. H. D. Sutherland, Dr. J. G. Sleeman and the Director of Tuberculosis.

Standing Committee on Public Health: Dr. Jose, Dr. Jungfer, Dr. McCartney, Dr. Viner Smith, Dr. Stokes, together with a representative from the Department of Public Health, with power to coopt.

Standing Committee on Hospitals: Dr. Jose, Dr. Elix, Dr. de Crespigny, Dr. J. R. Magarey, Dr. Mallen and the President Elect, with power to coopt.

The President, Immediate Past President, Vice-President, Honorary Treasurer and Honorary Medical Secretary are *ex-officio* members of all committees other than the Ethics and Standing Committees.

The President and Honorary Medical Secretary are *ex-officio* members of the Ethics and of all Standing Committees.

Attendances at Council and Committee Meetings.

Dr. C. G. Wilson attended eight meetings of Council in his capacity as local representative of The Editor of THE MEDICAL JOURNAL OF AUSTRALIA.

In addition the following Conferences have also been held during the year:

Conference with representatives of the Council and the South Australian Chamber of Manufactures, *re* certain aspects of the *Workers' Compensation Act*, July 30, 1959.

Meeting between Executive Committees of Council and the South Australian State Section, The Otolaryngological Society of Australia, November 2, 1959.

A further meeting of the Special Subcommittee (comprising representatives of all honorary medical staffs of teaching hospitals) in connexion with the administration of teaching hospitals in South Australia was held on April 4, 1960.

A meeting of the Building Committee was held on December 10, 1949, and a further meeting of members of the Memorial Hall Committee took place on August 13, 1959.

Monthly Scientific Meetings.

The following Scientific Meetings have been held during the year:

1959.—July 30: "Infant Welfare in South Australia". The following speakers took part: Dr. Helen Mayo, Dr. David Fearon and Dr. J. M. Last. September 1: clinical meeting, Repatriation General Hospital. September 24: "Symposium on Antibiotics". Speakers: Dr. K. V. Anderson (bacteriological aspects), Dr. Roger Angove (medical aspects), Dr. Ronald Hunter (surgical aspects). October 29: clinical meeting, The Queen Elizabeth Hospital, Woodville. November 26: The following subjects were presented: "Recent Advances in the Treatment of Tinea", Dr. Gordon Donald; "Carpal Tunnel Syndrome", Dr. Alan Campbell; "Recurrent Abortion", Dr. Ruth Heighway.

Attendances at Council and Committee Meetings.

	Council.	Salaries (Finance).	Scientific.	Parliamentary Bills.	Library.
BONNIN, N. J.	13	—	1	—	—
CHERRY, E. P.	11	—	—	—	—
DE CRESPIGNY, R. G. C. ..	11	—	1	1	3
DWYER, J. M.	13	2	1	1	3
GIBSON, G. T.	13	2	1	1	—
HALLORAN, N.	13	—	—	—	—
HECKER, R. ..	12	—	1	1	3
HEDDLE, R. C.	12	—	—	—	—
JEFFRIES, J. S.	9	—	—	—	—
JUNGFER, C. C.[1]	3	—	1	—	—
MAGAREY, I. S.	13	—	1	—	—
MALLEN, L. R.	8	—	—	1	—
OATEN, H. R.	13	2	—	—	—
RIEGER, C. O. R.	13	2	—	1	—
SLEEMAN, W. J.	10	—	—	—	—
Meetings to May 5, 1960 ..	13	2	1	1	3

[1] Leave of absence.

1960.—February 25: "Probing the Professors". The professional panel consisted of Professors H. N. Robson (Medicine), Professor R. P. Jepson (Surgery), Professor L. W. Cox (Obstetrics and Gynæcology), and Professor G. M. Maxwell (Child Health). March 31: clinical meeting, Adelaide Children's Hospital. April 21: "The Role of Heredity in Some Human Disorders". Speaker: Professor J. G. Bennetts. May 26: "Listerian Oration", delivered by Professor Lance Townsend of Melbourne, the subject being "High Blood Pressure and Pregnancy".

A number of interesting films has also been screened prior to some of the above meetings.

Additional Lectures.

1959.—July 22: by courtesy of Repatriation General Hospital, clinical meeting. August 25: by courtesy of Adelaide Children's Hospital, Swift Memorial Lectures. August 27: by courtesy of Adelaide Children's Hospital, Swift Memorial Lectures. October 14, by courtesy of Repatriation General Hospital, lecture. November 4: by courtesy of Repatriation General Hospital, film evening.

1960.—March 30: by courtesy of Repatriation General Hospital, film and discussion on paraplegia. April 27: by courtesy of Repatriation General Hospital, lecture on antibiotics.

Membership.

The membership of the Branch as at December 31, 1959, was 990. The number of student associate members at the same date was 48.

The deaths of the following members are recorded with regret: Dr. Gilbert Brown, Dr. Kenneth Fry, Dr. C. F. Drew and Dr. P. Cockburn.

Appointments and Nominations.

Advisory Council on Health and Medical Services: Dr. L. R. Mallen.

B.M.A. Services Limited: Dr. R. G. C. de Crespigny, Dr. R. M. Glynn, Dr. B. S. Hanson, Dr. L. R. Mallen and Dr. C. O. F. Rieger (Chairman).

British Medical Association, Annual Representative Meeting, Torquay, 1960: Sir Phillip Messent, and Dr. W. D. Ackland Horman.

Central Council of the Association, London: Dr. Myles Formby.

Chiropody Board of South Australia: Dr. Neville Wilson.

Dental Board of South Australia: Dr. Graham Bennett.

Federal Council of the British Medical Association in Australia: Dr. L. R. Mallen and Dr. C. O. F. Rieger.

Royal Flying Doctor Service of Australia (S.A. State Section): Dr. J. M. Dwyer.

Florence Nightingale Memorial Committee of Australia (S.A. Branch): Dr. Dorothy Adams and Dr. Mary Walker.

Medical Board of South Australia: Dr. C. O. F. Rieger.

Mothers and Babies' Health Association: Dr. Nevill Bickford.

Nurses' Board of South Australia: Dr. Thorold Grant.

Old People's Welfare Council of South Australia: Dr. C. Duguid and Dr. J. G. Sleeman.

Rehabilitation Standing Committee (Departmental): Dr. J. R. Barbour.

S.A. Association for Mental Health Inc. (Council): Dr. S. B. Forgan.

"The Medical Journal of Australia" (Editor's Representative for South Australia): Dr. C. G. Wilson.

The St. John Ambulance Brigade (S.A. District): Dr. H. H. Hurst (Deputy Commissioner).

University Post-Graduate Committee in Medicine: Dr. F. L. Thyer, Dr. Robert Hecker and the President *ex officio*.

World Medical Association: Dr. L. R. Mallen (Chairman of Council).

Sections Formed for Special Knowledge.

Australian Association of Anæsthetists (South Australian Branch).

The South Australian Branch now comprises 46 members. At the annual general meeting in February the following office-bearers were elected: Chairman, Dr. M. C. Newland; Chairman-Elect, Dr. H. J. Ellis; Past Chairman, Dr. J. A. Ferris; Honorary Treasurer, Dr. M. J. W. Sando; Honorary Secretary, Dr. D. P. Dineen.

Six meetings were held during the year, and as well as papers by local members, variety was provided by films through the courtesy of I.C.I. Dr. J. Stace made available tape recordings of proceedings of American Anæsthetic Association meetings. The medium has high potential for the interesting exchange of information and experience.

Two guest lecturers were invited; Dr. N. F. Cass of Melbourne, and Professor Mushin of Cardiff, Wales, was again a most successful overseas visitor.

It was with great regret that the Society records the death of Dr. Gilbert Brown during the year. Dr. Brown will long be remembered for his contributions to anæsthesia and to this Society in particular.

Section of Clinical Medicine.

There are 40 financial members of the section, an increase of one over last year.

At the annual general meeting held on June 16, 1959, the following officers were elected: Chairman, Dr. R. F. West; Honorary Secretary and Treasurer, Dr. S. C. Milazzo; Committee, Dr. T. H. Beare, Dr. J. L. Waddy, Dr. G. Ey and Dr. J. Lawrence. Subsequently, the Committee coopted Dr. S. Posen, in view of the temporary absence overseas of Dr. Waddy and Dr. Lawrence.

Clinical meetings were held at the Chest Clinic in June, September and November, 1959, and in March, 1960, the average attendance being 37 members.

At the meetings a wide range of cases was presented from all the major hospitals, including the Repatriation General, Adelaide Children's and Northfield Infectious Hospitals, as well as from the Queen Elizabeth and Royal Adelaide Hospitals.

It has been felt that the Section has a valuable function in bringing together clinicians from the various hospitals, especially in view of the increasing tendency of the hospitals to arrange their own clinical meetings.

The lack of presentations from doctors not associated with hospitals is unfortunate, and the Committee continues to hope that a better response to the open invitation (published in the monthly circular) to show cases will be forthcoming.

It is also hoped that the essentially clinical nature of the meetings will be preserved by patients being presented "in the flesh" whenever possible.

The British Medical Association (Section of Clinical Medicine) Prize was won by Miss Carlien Lorraine Fahy.

The Ophthalmological Society of Australia (South Australian State Section).

During the year it was decided to disband the Section of Ophthalmology and to form a State Section of the Ophthalmological Society of Australia. As practically all South Australian ophthalmologists are members of the Ophthalmological Society of Australia, this move was made with the view to coordinating the activities and interests of members in this State with those of other States. Any interested members of the British Medical Association interested in ophthalmology are welcome to attend the various meetings which are held during the year.

The following meetings were held since July, 1959:

1959.—July 14. a paper entitled "Thrombosis of the Carotid Arteries" was presented by Professor Jepson. September 29: inaugural meeting of the South Australian State Section of the Ophthalmological Society of Australia. The following officers were elected: Chairman: Dr. D. O. Crompton; Vice-Chairman: Dr. T. L. McLarty; Honorary Secretary and Treasurer: Dr. M. C. Moore. December 8: clinical meeting at which a number of cases were presented by members. Dr. M. C. Moore reported on the results of a survey carried out by Dr. H. A. Handley, Dr. D. O. Tonkin, Dr. P. J. Stobie and himself on the vision of pre-school kindergarten scholars. Resulting from this survey, recommendations were made to the Kindergarten Union concerning the examination of such children.

1960.—March 8: clinical cases were shown, and two films entitled "Ocular Infections" and "Cardiac Arrest" were screened. April 28-May 4: Together with radiotherapists and members of the Oto-Laryngological Society, members attended lectures by Dr. M. Lederman, visiting Cancer Professor, on "Radiotherapy of Benign and Malignant Ocular Conditions.

The average attendance at these meetings was 13.

The Oto-Laryngological Society of Australia (South Australian State Section).

The annual general meeting of the Oto-Laryngological Society of Australia, South Australian State Section, was held on March 17, 1959. The following officers were elected: Chairman, Dr. R. H. von der Borch; Vice-Chairman, Dr. P. G. Jay; Committeeman, Dr. R. G. Plummer; State Representative, Dr. O. S. deB. Cocks; Secretary-Treasurer, Dr. R. E. Gristwood.

Ordinary general meetings were held on May 19, July 14, and October 13, 1959.

Dr. A. J. Hakendorf presented a paper on the "Dermatological Aspects of Otitis Externa", illustrated by coloured slides, at the July meeting, and Dr. D. A. Simpson gave a paper on "Vertigo as a Symptom of Intracranial Neoplasms" in October.

Special general meetings were held on March 17 and 24, to consider a plan for the management of deaf and hard-of-hearing children in South Australia.

A further extraordinary meeting took place on December 8, to consider the available teaching facilities in oto-laryngology in South Australia, and also to consider the problem of standardization in audiometry.

The average attendance at meetings was eight.

Affiliated Local Associations of Members.

Northern Medical Association.

During the year four meetings were held, as follows:

1959.—August 2, at Blyth: speakers were Professors L. W. Cox, on "Toxæmia of Pregnancy", and Mr. I. B. Jose, on "Limp in Childhood". Seven members were present. November 1, at Gladstone: speakers were Dr. G. W. Verco, on "Urinary Tract Conditions with Special Relation to Pædiatrics", and Dr. R. M. MacIntosh on "Menstrual Irregularities". Six members were present.

1960.—February 7, at Clare: speakers were Dr. R. A. Burston, on "Present Views on the Management of Diabetes

Mellitus", and Mr. J. Howard Brown, on "Carcinoma of the Bronchus', Mr. G. G. Wyllie, on "Management of the Undescended Testes and Other Penile Deformities—Enuresis", and Dr. Howard Linn, on "Diagnosis and Treatment of Annular Skin Diseases". Seven members were present. May 7, at Port Pirie: at this meeting, Dr. R. F. Matters showed a film demonstrating the methods of, and reasons for taking routine cervical smears for cytological examination in women over 35 years of age. The film was followed by a lecture by Professor M. Lederman, of the Royal Marsden Hospital, London, on "Radiotherapy in the Treatment of Carcinoma with Special Reference to Gynæcology and Oto-Rhino-Laryngology". This meeting was attended by twelve members, and was preceded by dinner. Office-bearers for the year were: President, Dr. V. W. Potter; Vice-President, Dr. R. T. Davidson; Honorary Secretary, Dr. L. J. Daly.

Upper Murray Medical Association.

Three post-graduate week-ends were arranged with the Post-Graduate Committee in Medicine. These were: May, 1959, Waikerie: visiting lecturers were Dr. J. M. McPhie, Dr. A. A. Tye and Dr. J. S. Skipper; August, 1959, Loxton: visiting lecturers were Dr. R. G. Plummer, Dr. Ronald Hunter and Dr. I. M. H. Camens; November, 1959, Barmera: visiting lecturers were Dr. R. F. West, Dr. Mervyn Smith and Dr. R. A. Thatcher.

In addition, three local meetings have also been held at Renmark, Berri and Loxton. These were held in April and September, 1959, and April, 1960, respectively.

There are eleven members of the Association, the average attendance being eight.

The following officers have been elected for the ensuing year: President, Dr. B. J. Downing; Vice-President, Dr. K. E. Evans; Honorary Secretary, Dr. T. G. Kohler.

Salisbury and Elizabeth Medical Association.

Following the opening of the Lyell McEwin Hospital in May, 1959, a new local association of members was formed in the Salisbury and Elizabeth District. Affiliation with the State Branch of the British Medical Association followed.

The following were elected to the Committee: President, Dr. F. E. Trembath; Honorary Secretary-Treasurer, Dr. B. T. J. Newland; Committee Members, Dr. E. D. Richards and Dr. V. Hart.

A scheme of group ownership of surgical instruments and expendible equipment, such as catgut, anæsthetic agents, gloves, needles, etc., was introduced. Individual practices pay a monthly account based on the number of operations and anæsthetics performed. This method eliminates loss of time by nursing staff looking after instruments belonging to individual doctors. The method has worked well for twelve months, and is appreciated by visiting surgeons, who need not bring any equipment with them. The following guest speakers have addressed members: Dr. C. C. Jungfer gave an interesting address on trends in medical practice in Australia and abroad; Dr. O. W. Leitch spoke on "Injuries to the Hand"; Dr. L. Male spoke on "Rehabilitation and Social Services". Dr. H. Gilmore demonstrated a cardiac defibrillator, and spoke on treatment of cardiac emergencies during surgical operations.

The Association wishes to record thanks to these speakers. A considerable number of consultants, both medical and surgical, have visited the hospital. Their help has enabled local practitioners to treat many patients in their own area.

South-Eastern Medical Association.

Five meetings were held during the year.

1959.—August 8: clinical meeting at Mt. Gambier followed by dinner, after which Dr. A. W. Burnell spoke on "Physical Medicine". October 31: clinical meeting at Millicent, followed by a dinner, after which Mr. G. Wyllie spoke on "Do's and Don'ts in Pædiatric Surgery".

1960.—February 27: clinical meeting at Mt. Gambier followed by dinner, after which Dr. B. S. Hanson spoke on "Radiotherapy". April 30: the annual general meeting was held at Naracoorte, and after dinner Professor M. Lederman spoke on "Diagnosis and Management of Carcinoma of the Head and Neck and Uterus". At this meeting the following were elected as officers for the ensuing year; President, Dr. H. B. Holmes; Vice-President, Dr. D. K. Kumnick; Secretary-Treasurer, Dr. C. A. Leeson; Committee, Dr. K. J. Wilson, Dr. D. K. Kumnick and Dr. J. E. Dunn.

The annual post-graduate refresher course was held at Mt. Gambier on July 12, 1959. Visiting speakers were: Mr. D. N. Robinson, "Injuries of the Hand"; Mr. I. B. Jose, "Chronic Urinary Tract Infection. Secondary Carcinoma. Prostatism"; Professor Farquharson, "Emotional Manifestation in Patients with Structural Disease".

There are 36 financial members of the Association, and the average attendance at meetings, excluding visitors, has been 25.

Yorke Peninsula Medical Association.

In the year 1959, the Association meetings took the form of week-end courses arranged by the Post-Graduate Committee in Medicine. At these meetings, the Committee met and dealt with current matters. The officers for the year were: President, Dr. P. Reilly, of Minlaton; Honorary Secretary, Dr. G. J. Smibert, of Wallaroo.

The following week-end courses were held:

1959.—March 1: Professor Jepson, Professor of Surgery, discussion of cases presented by members; Dr. J. V. Gordon, "Headache". March 31: Dr. J. L. Hayward, "Management of Asthma"; Dr. A. D. Byrne, "Prolapse and Stress Incontinence'. August 30: Dr. A. G. Campbell, "Cholecystectomy and its Complications"; Dr. F. L. Thyer and Dr. E. F. Gartrell, "Coronary Sclerosis and Consideration of Electrocardiograms". November 29: Dr. L. L. Hoare, "Vehicular Accidents and the Treatment of Head Injuries"; Dr. L. O. S. Poidevin, "Surgical Induction and its Dangers".

At the annual general meeting, held on March 6, 1960, the following officers were elected for the ensuing year: President: Dr. W. I. Seith; Honorary Secretary: Dr. J. S. Flett.

Organization of the Medical Profession in Australia.

Members will no doubt have noticed from the published report that at the last meeting of the Federal Council held on February 26, 1960, the following resolutions were adopted:

(i) That the Federal Council, having received the unanimous approval of the Branches, will now proceed with the formation of an Australian Medical Association,

(ii) That the Federal Council appoint a Committee to consider ways and means of forming an Australian Medical Association, to consult the Association's legal adviser and to produce a draft constitution for submission to any consideration by the Federal Council at its next meeting, if possible.

(iii) That the Committee referred to in the previous resolution should be a Steering Committee with authority to have discussions with the Branches of the British Medical Association in Australia, with any other medical bodies which in the opinion of the Committee should be consulted, and that the draft constitution to be produced be a skeleton draft for consideration by the Federal Council.

The South Australian Branch has already forwarded its suggestions in connexion with the proposed constitution of an Australian Medical Association to the Federal Council, and the matter will be further considered at the next meeting, which will be held in Adelaide on August 5 next.

In reply to a personal letter from the President of the Federal Council, the Chairman of Council (Dr. Wand) has indicated that the move to form a Medical Association of Australia is not entirely unexpected. He states that no one in the United Kingdom would wish to do anything which might weaken the traditional bonds which have united the professions in the two countries, and endorses the wish that these bonds will be maintained by means of affiliation with the Parent Association.

There appears to be no doubt that the medical profession in Australia will eventually follow the pattern set by the Canadian and South African Medical Associations respectively, both of whom are affiliated with the British Medical Association.

At a meeting of the Branch Council held on June 2, 1960, a recommendation was received from the Victorian Branch, which suggested that in view of the inevitable delay in completing the formation of the Australian Medical Association, and in view of the fact that this delay costs the medical profession of Australia approximately £26,000 *per annum*, steps be taken to form the new Association forthwith, and that the Federal Council act as provisional executive body with a simple constitution which can be modified and finalized at some future date.

The Council supported this recommendation, which it is envisaged will necessitate special meetings of the Federal

Council. Before any form of draft Constitution is adopted, however, this will require to be submitted to, and approved by, members of the Branch at an extraordinary meeting called for that purpose. All sections of the medical profession may therefore be assured that an adequate opportunity will be given to them of stating their views on the matter.

Australasian Medical Congress—Eleventh Session.

During the year a certain amount of preliminary work has been undertaken with a view to making plans to hold the next Session of Congress in Adelaide from May 19 to 25, 1962.

The Executive Committee has met on several occasions, and a chairman of the numerous subcommittees has been appointed. In addition, on April 5 last, a meeting of the wives of members of the executive was held for the purpose of discussing arrangements covering the entertainment and social aspects of Congress, and appointments as conveners of the various ladies' committees have been made.

Pharmaceutical Benefits Act.

The implementation by the Government of an extended Pharmaceutical Benefits Scheme on March 1, 1960, has been the cause of much dissatisfaction on the part of many members due, in the main, to the fact that these proposals were implemented without the organized medical profession first being given an opportunity to study them. On April 27, 1960, a communication was forwarded to all country local associations and metropolitan groups, informing them that there was a general feeling of disquiet with many aspects of the Act in its new form, and that although the Federal Council had urged the Government to delay the provisions for a period of two months after the terms and conditions were known, to allow time for further consideration, the Minister had rejected this request. The Groups were advised of the points which should be borne in mind in any future deliberations.

In the light of experience since the implementation of the new provisions, the fears of many members that this would interfere with their prescribing freedom have not been allayed. Although various aspects of the new scheme may continue to be subject to considerable controversy, it will be agreed that the profession must avoid taking any precipitate action which could be interpreted as ill-conceived. To gain public sympathy for any stand which may be taken, it will have to be shown that our opposition to it does not stem from sectional interests, but that the measure works to the detriment of the patient, not only financially, but especially by its restrictive character and a tendency to enforce bad medical standards. The public relations aspect of the problem will, therefore, need to be given careful consideration; but in view of the approach which was made by the Council to the various country local associations and metropolitan groups, it is likely that the subsequent views of these members will indicate the action to be taken, rather than by means of a plebiscite of all members. Members will be advised of further developments.

Memorial Hall.

A very notable occasion in the history of the Branch was the opening of the Memorial Hall by the Honourable the Premier of South Australia (Sir Thomas Playford) on Wednesday, May 11, 1960, at 8.30 p.m., in the presence of a number of distinguished local and interstate guests.

It is interesting to recall that the move to provide a hall for the Branch first began on December 13, 1913, when a group of interested doctors took steps to form the British Medical Hall Company Limited with this in view. The first two objects of this company were:

> 1. To purchase the freehold land and buildings known as Lady Colton Hall, Hindmarsh Square, Adelaide . . . .
>
> 2. To permit the said land and buildings or any part thereof, or other land or buildings purchased or otherwise acquired by the Company to be used by the South Australian Branch of the British Medical Association, on such terms as the Company shall think fit, and at a nominal rental, or rent-free, and for any other purpose which the Branch or its Council may, in its or their absolute discretion, deem necessary, proper or desirable in the interests of the Branch.

The Company was formed for the purpose of conducting a medical agency, and to engage in this and other income-producing activities with members of the medical profession on behalf of the Branch, whose rules and by-laws did not permit it to do this. The necessary finance to purchase the Hindmarsh Square property was raised by issuing shares in the Company to doctors who contributed (only registered medical practitioners were permitted to hold shares), and the property was later bought and the Lady Colton Hall used for scientific meeting, lectures, etc., by the Branch for a number of years following its acquisition.

It was not realized that this property had the disadvantage of being situated in close proximity to several tramlines, and the resultant noise associated with the horse-drawn tramcars of those days was such that the concentration of both speakers and audience quickly became disturbed. For this and other reasons, the property became unsuitable and was later sold at a profit, and a large part of the proceeds invested in a (then) vacant block of land situated on North Terrace, next to the Electricity Trust premises, on which Elizabeth House now stands. This block was held by the Company for a number of years, including those of the second World War, and was, in turn, sold in May, 1949, at a greatly enhanced value, in favour of a site situated in Kermode Street, North Adelaide. This latter site offered ease of access, with right-of-way to the property from King William Road, with no attendant parking problems.

Whilst still holding this site, the present property situated at 80 Brougham Place, North Adelaide, became available, and quickly realizing the advantages this possessed over the Kermode Street site, steps were taken by the Company to purchase this in 1951 at auction for the sum of £11,500. It soon became evident that the move to the present site was undoubtedly the most advantageous, both financially and geographically, that the Company had ever made in the whole of its previous existence.

A further amount of £10,500 was expended in alterations and additions to the property, so as to provide suitable office accommodation for the Branch and its secretariat, in addition to three professional suites. The Kermode Street property was later sold advantageously.

On December 18, 1959, the Hall Company was placed in voluntary liquidation and the assets of the Company (including the Brougham Place property) were placed in the name of the Branch. This was made possible by the Branch acquiring either by gift or purchase of all shares not owned by it in the Company. A special Building Committee under the chairmanship of Sir Henry Newland — who, incidentally, had also been one of the original founders of the Hall Company and its first honorary secretary, then later chairman for many years—was appointed to discuss with the architect the provision of badly-needed office accommodation and the erection of a Memorial Hall. A tender for the sum of £41,556 was accepted and the project, which commenced on July 6, 1959, was completed at the end of March, 1960.

It was indeed fitting that the history of these events should be related to those present at the official opening ceremony by Sir Henry Newland, who had done so much to bring the building plans of the Branch to fruition. Now, just over 36 years since the Hindmarsh Square property was disposed of, the Branch again has a hall of its own. This has been dedicated to the memory of the medical officers who lost their lives in the first and second World Wars.

It is to be regretted that the Building Fund Appeal has not met with the support which was originally anticipated, the total amount subscribed to date being £4909 0s. 9d. with promises for a further £954 3s. The total cost of the project will not be known until the submission of final accounts, although a ceiling of £50,000 was placed upon the project before building operations commenced. Reports so far indicate that the total cost, including furnishings, will be within this figure. At the same time, the Building Fund will remain open for receipt of donations, as all contributions will lessen the actual amount requiring to be borrowed and a valuable saving in interest charges will be effected.

The Queen Elizabeth Hospital.

It may perhaps not be recognized generally by members that a unique experiment is in the process of development at the Queen Elizabeth Hospital, Woodville, in which a general practitioner floor, comprising fifty beds, has been incorporated. These are available for the use of the private patients of practitioners throughout the State. They are not open to any practitioner who holds the office of honorary physician or surgeon, or honorary assistant physician or surgeon, as the case may be, on the staff of a hospital. It is believed that no parallel situation exists in any other part of the Commonwealth, where a separate floor in a teaching hospital has been set aside solely for the treatment of private patients of practitioners.

SOUTH AUSTRALIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION INCORPORATED.

Balance Sheet as at December 31, 1959.

LIABILITIES.	£ s. d.	£ s. d.
Sundry Creditors		2,846 0 3
Mortgage		8,000 0 0
General Fund:		
Balance, January 1, 1959	12,695 5 11	
Net Surplus for year	635 11 2	
	13,330 17 1	
British Medical Hall Liquidation Net	23,606 17 4	
Transfer from Building Fund	10,712 6 0	
		47,650 0 5
		£58,496 0 8

ASSETS.	£ s. d.	£ s. d.
Fixed Assets:		
Newland House (subject to mortgage)	27,824 7 4	
Memorial Hall	17,932 15 2	
Furniture and Plant (at cost less Depreciation)	4,624 5 7	
Delineascope and Projector (at cost less depreciation)	3 18 2	
Lister Medal (at cost less depreciation)	2 0 0	
		50,387 6 1
Current Assets:		
Australasian Medical Publishing Company Shares	4,030 0 0	
Advances not converted	37 3 1	
British Medical Association Services Limited Shares	500 0 0	
Stock on Hand	152 16 6	
Sundry Debtors	1,722 7 0	
Cash at Bankers:		
National Bank of Australia Ltd.	105 18 11	
Commonwealth Savings Bank	171 5 1	
Savings Bank of South Australia	403 19 9	
Trust Account, Commonwealth Savings Bank	950 0 0	
Savings Bank of South Australia Library Account	2 0 0	
Cash on Hand	33 4 3	
		8,108 14 7
		£58,496 0 8

Special Funds and Investments.

	£ s. d.	£ s. d.
Listerian Oration Fund:		
Balance, January 1, 1959	364 19 9	
Add Net Income	9 10 7	
	374 10 4	
Less Oration Expenses	39 8 0	
		335 2 4
Frank S. Hone Memorial Fund:		
Balance, January 1, 1959	745 10 9	
Add Net Income	33 15 6	
	779 6 3	
Less Prizes	31 10 0	
		747 16 3
Trent Champion de Crespigny Memorial Fund:		
Balance, January 1, 1959	304 3 1	
Add Net Income	9 11 2	
		313 14 3
Building Fund:		
Balance, January 1, 1959	4,348 13 11	
Add Interest and Donations	7,767 16 11	
	12,116 10 10	
Less Sundry Expenditure	10,712 6 0	
		1,404 4 10
		£2,800 17 8

	£ s. d.	£ s. d.
Listerian Oration Investments:		
E.T.S.A. Debentures	310 0 0	
Savings Bank of South Australia	25 2 4	
		335 2 4
Frank S. Hone Memorial Fund Investments:		
Commonwealth Bonds	50 0 0	
South Australian Gas Co. Debentures	300 0 0	
E.T.S.A. Debentures	250 0 0	
Savings Bank of South Australia	147 16 3	
		747 16 3
Trent Champion de Crespigny Memorial Fund:		
Savings Bank of South Australia		313 14 3
Building Fund:		
Commonwealth Bond, 1960	200 0 0	
Savings Bank of South Australia	417 16 1	
Commonwealth Savings Bank	466 3 9	
Commonwealth Savings Bank General Fund	320 5 0	
		1,404 4 10
		£2,800 17 8

F. C. W. DOBBIE, Secretary.

J. M. DWYER, Hon. Treasurer.

We hereby report that we have examined the Books and Accounts of the South Australian Branch of the British Medical Association Incorporated for the year ended December 31, 1959. In our opinion the above Balance Sheet, together with the Statements of the Special Funds, are properly drawn up so as to exhibit a true and correct view of the affairs of the Branch, as at the above date, according to the best of our information, the explanations given to us, and as shown by the books produced.

Adelaide, May 23, 1960.

ANNELLS, TILLEY, HUNWICK AND CO.
Chartered Accountants.

Income and Expenditure Account for Year Ended December 31, 1959.

1958		£	s.	d.
15.	To Audit Fee	15	15	0
191	„ Depreciation	212	15	6
149	„ Entertainment	149	13	9
650	„ General Office Expenses	1,103	10	10
—	„ Interest on Mortgage	50	0	0
9	„ Legal Expenses	193	1	6
466	„ Postages	543	18	0
2,106	„ Secretariat	2,203	15	5
573	„ Printing and Stationery	551	19	11
48	„ Telephone	104	19	11
1,141	„ Net Surplus for Year	635	11	2
		£5,765	1	0

1958		£	s.	d.	£	s.	d.
	By Gross Subscriptions:						
7,177	City	7,586	12	3			
2,146	Country	2,178	15	3			
45	Students	81	11	0			
					9,846	18	6
	Less Deductions:						
1,444	B.M.A., London	1,477	19	0			
459	Medical Journal of Australia	466	2	6			
939	Library	982	16	0			
1,118	Federal Council	1,155	0	0			
					4,081	17	6
	Net Subscriptions				5,765	1	0
					£5,765	1	0

A conference between the Medical Superintendent and the Administrator of the hospital, who met the President of the Branch and representatives from the Woodville and Port Adelaide General Practitioner Group and the South Australian Faculty of the Australian College of General Practitioners, was held on February 11, 1960, to discuss the question of the administration of this floor, including the proposed admitting and other procedure. General agreement was reached on all points, and the first of the beds on this floor became available for the reception of patients on March 8, 1960.

Direct contact by general practitioners with a teaching hospital under a system such as this should be the means of maintaining a high standard of medical care for the public which the hospital is designed to serve.

There is no reason why this move cannot serve as a precedent in the development of other regional hospitals. All sections of the medical profession must therefore do their utmost to ensure that the experiment is a success so that it can be shown that a system under which a general practitioner floor is incorporated in a public hospital can be made to work successfully, providing all sections of the profession are determined to make it do so.

Medical Certification.

This report would not be complete without the usual reference to medical certification. The Council believes that the efforts which it has made in the past to educate members to a better sense of their responsibilities are slowly but surely beginning to achieve some result. Whilst the odd complaint or two still comes forward from the Employers' Federation, the volume of these is steadily decreasing. These complaints would probably disappear entirely if members would refrain from issuing a certificate unless they have actually attended a patient during an illness for which sick leave is claimed.

Honorary Life Membership.

The Council extends its congratulations to the following members, all of whom completed 50 years' continuous membership during the year and thus became Honorary Life Members of the British Medical Association: Dr. Edgar Brown, Dr. M. Erichsen, Dr. D. Parkhouse, Dr. A. Sandison.

Federal Council of the British Medical Association in Australia.

The Federal Council met in Brisbane on September 26, 1959, and in Melbourne on February 26, 1960. Reports of these meetings were later published in THE MEDICAL JOURNAL OF AUSTRALIA.

Frank S. Hone Memorial Fund.

The prize which was awarded on the results of the 1959 examinations for the degrees of bachelor of medicine and bachelor of surgery was won by Carlien Lorraine Fahy.

Honours and Awards.

The Council extends its congratulations to the Vice-President (Dr. R. G. C. de Crespigny), who was awarded the O.B.E. in the New Year Honours List, and to Dr. H. G. Prest, who has been admitted to the Grade of Officer in the Venerable Order of St. John of Jerusalem.

G. T. GIBSON,
President.

FINANCIAL STATEMENT.

The financial statement was presented by the Honorary Treasurer, Dr. J. M. Dwyer, and adopted.

ELECTION OF OFFICE-BEARERS.

There being no other nomination, Dr. Gibson declared Dr. R. G. C. de Crespigny elected to the office of President for the ensuing year, and invested him with the badge of office. Dr. de Crespigny thanked the members for his election.

Dr. de Crespigny then announced the following office-bearers for the ensuing year:

Vice-President: Dr. R. M. Glynn.

Honorary Treasurer: Dr. J. M. Dwyer.

Honorary Medical Secretary: Dr. R. Britten Jones.

Members of Council: Group A (metropolitan), Dr. G. A. Hodgson, Dr. H. W. Linn, Dr. R. S. Wilkinson. Group B (country), Dr. H. B. Holmes, Mount Gambier. It was passed as a recommendation to the Council taking office on July 1, 1960, that Dr. W. J. Sleeman, of Renmark, be invited to fill for a further 12 months the vacancy caused by a failure to elect a country member of Council in June, 1959.

Messrs. Annells, Tilley, Hunwick and Company were appointed auditors for the ensuing year.

PRESENTATION OF SECTION OF CLINICAL MEDICINE PRIZE.

Dr. R. A. A. Pellew, chairman of the Section of Clinical Medicine, presented the winner of the Section's prize for 1959, Dr. Carlien Fahy, to Dr. Gibson. Dr. Gibson presented Dr. Fahy with a certificate of the award.

RETIRING MEMBERS OF COUNCIL.

A vote of thanks was passed to the retiring members of Council, Dr. N. J. Bonnin, Dr. N. Halloran, Dr. R. C. Heddle, Dr. J. S. Jeffries, Dr. C. C. Jungfer and Dr. R. Hecker.

RETIRING PRESIDENT'S ADDRESS.

Dr. G. T. Gibson delivered his retiring president's address (see page 321), after which a vote of thanks was passed to him.

ANNUAL MEETING OF THE BRITISH MEDICAL ASSOCIATION, AUCKLAND, FEBRUARY, 1961.

THE 129th annual meeting of the British Medical Association is to be held in Auckland in 1961, as was previously announced. The scientific programme extends from Monday, February 6, to Friday, February 10; but during the preceding week-end there will be time allotted for registration and certain social activities. The following is an outline of events.

Monday, January 30: Auckland Anniversary Regatta.

Friday, February 3: Registration, overseas visitors; visit to Kinleith, the timber, board and paper mill of New Zealand Forest Products, near Tokoroa.

Saturday, February 4: Registration, sport and social activities.

Sunday, February 5: morning, official church services, Church of England and Roman Catholic; afternoon, garden party at the home of Sir Frank and Lady Mappin; evening, theatre—film première at the Odeon Theatre.

Monday, February 6: 9 a.m., registration and opening of pharmaceutical and scientific exhibitions by Sir Douglas Robb; 11 a.m., opening of conference by His Excellency the Governor-General; 11.30 a.m., inaugural address, Professor E. Sayers (Dean of Otago University Medical School); 12.30 p.m., conference photograph; 2.30 p.m., plenary session, "Maintaining the Health of the Middle-Aged Executive"; 8 p.m., annual general meeting of the British Medical Association—presidential induction, address and reception.

Tuesday, February 7: 8 a.m., Christian Medical Fellowship breakfast; 9.30 a.m., day visit to Wenderholme Beach and Waiwera Hot Springs; 9.30 a.m., plenary session, "The General Surgical, Medical and Psychological Problems of Pregnancy"; 11.30 a.m., round table conference, "The Present Position of Steroid Therapy", and sectional meetings; 2.15 p.m., sectional meetings; 3.30 p.m., sectional meetings; 7.30 p.m., conference dinner at the Australian Pavilion, Epsom Showgrounds.

Wednesday, February 8: 9.30 a.m., plenary session, "Chemotherapy in Cancer", 11.30 a.m., launch trip on Waitemata Harbour.

Thursday, February 9: 9.30 a.m., plenary session, "Iso-Immunization—A New Concept of Disease"; 11.30 a.m., round table conference on "Ulcerative Colitis", and sectional meetings; 2.15 p.m., sectional meetings; 4.30 p.m., afternoon garden reception by the British Medical Association at Ellerslie Racecourse.

Friday, February 10: 9.30 a.m., visit to Puketutu Island on Manakau Harbour; 9.30 a.m., plenary session, "Mental Health in the Community"; 11.30 a.m., round table conference, "Thyroid Disease", and sectional meetings; 2.15 p.m., sectional meetings; 3.45 p.m., annual meeting of the British Medical Association (New Zealand Branch); 4.45 p.m., War Memorial Oration at the Auckland Grammar School—speaker, Mr. Kenneth MacCormick, C.B., C.B.E., D.S.O.; 9 p.m., conference ball in the Town Hall.

After the conference, it is expected that overseas visitors will take the opportunity of visiting other parts of New Zealand. Visitors are asked to make their own travel arrangements through recognized travel agencies; but the Travel Commissioners in Sydney (14 Martin Place) and Melbourne (428 Collins Street) will be glad to advise and book tours. Throughout New Zealand members of the British Medical Association have offered to act as hosts, and if visitors can indicate their plans and interests, the conference committee will arrange hospitality in each centre they pass through. The Southland Division of the New Zealand Branch has offered to arrange a comprehensive four-day holiday in Fiordland, commencing on Thursday, February 16, and comprising the following: (i) Milford Track—a four-day trip with reasonable hut accommodation; (ii) Doubtful Sound Track—a three-day trip requiring greater fitness and less endowed with scenery than (i); (iii) boat excursions on Lakes Te Anau and Manapouri; (iv) climbing parties in the Homer tunnel area for the very fit; (v) lake and river picnics; (vi) car trips to the Eglington and Hollyford Valleys and through to Milford Sound; (vii) flights over Fiordland or to Queenstown; (viii) a film and supper evening on the Saturday night; colour slides and films of the area will be shown. The Southland Division will arrange for private hospitality during this period; but if visitors wish hotel accommodation and bookings on the two main walking tracks, early application will be necessary.

Out of the Past.

A UNIVERSITY LECTURER ON PHARMACY.[1]

[From the *Australasian Medical Gazette*, December, 1901.]

THE University of Adelaide is the first to appoint a Lecturer in Pharmacy for pharmaceutical students in Australasia. When pharmaceutical education was first seriously considered, the Pharmacy Board of Victoria made vigorous efforts to persuade the Melbourne University to provide the necessary instruction: this was refused. Many years after, the South Australian Board found it possible to induce the University of Adelaide to make provision for the teaching of pharmaceutical students in the purely scientific subjects of Chemistry, Botany and Materia Medica, and now, owing largely to the persistence and enthusiasm of one or two men the University has consented to appoint a Lecturer on Pharmacy on the nomination of the Pharmaceutical Society.

The New South Wales pharmacists have lately made arrangements with the Sydney University for work of this kind, and are agitating for the provision of regular pharmacy lectures.

[1] From the original in the Mitchell Library, Sydney.

Correspondence.

IT HAPPENED ONE NIGHT.

SIR: In reply to the letters in your correspondence column following the article "It Happened One Night", by myself and A. T. Bath (MEDICAL JOURNAL OF AUSTRALIA, May 28, 1960), I appreciate the remarks of Dr. Clive Sands of Canberra. I regret that I did not see his article on the south-west wind until after I had corrected the proofs of my own. As Dr. Sands says in the article, the possibility of temperature inversions was discussed, affecting the periodicity of hay fever in Sydney in allergic individuals.

H. A. and E. R. Heise[1] quote the effect of temperature inversion on the distribution of pollen and mould spores, and pointed out, *inter alia*, that the inversion which lasted five days in Donora, Pennsylvania, in October, 1948, caused 20 deaths and the illness of 5910 persons, 43% of the population.

The weather conditions that caused this tragic circumstance are described by L. Machta in the *Public Health Reports*[2] of the U.S. Department of Health Education and Welfare, and were caused by two of the three types of inversion discussed later in this letter, one of which was the type I described in my original paper as occurring for two days over Brisbane on April 20, 1959.

Dr. Rogers of Mt. Isa asked definite questions, to which I will give the answers (in collaboration with my co-author) in the order in which he asked them.

1. At what hour did the temperature inversion move to a lower height on the night of April 20, 1959? In this case the inversion started at sunset and the effect was progressive, reaching a maximum in the early hours of the following morning about sunrise. I will discuss this type of inversion later in the letter.

2. The graphs are labelled "altitude over Brisbane". It is quite reasonable to assume the increase in atmosphere pollution applied to the wide area quoted, because Brisbane being an industrial city and the inversion occurring on a week day would naturally cause pollution of the air over that wide area. Furthermore, pollens and dust which have been distributed in that area over the previous day would all be concentrated very markedly over that area during the night.

3. Dr. Rogers takes me to task for the words "at exactly 11.45 p.m.". I quote from a night report book where the entry was made by the sister on duty as 11.45 p.m., and it was pointed out to me by the staff in that ward next morning. It is not likely that Dr. Rogers' surmise was correct, as the ward concerned was an intermediate ward and the patients were widely separated in different rooms in the ward.

4. If Dr. Rogers had read my article in the first place he would have noticed that my observations went on over a period of some 12 years and this was the first night on which there were no variables. Retrospective survey would not be easy, as I explained in my covering letter to the article to the Editor of this Journal, as it required a very great amount of time on the part of the Commonwealth Meteorological Bureau to check the records time after time while we were endeavouring to get to the cause. Continuing the investigations is the only feasible method. I have continued my work on this subject, as is mentioned later in this letter.

In question 5, I feel that Dr. Rogers was just being facetious. His next statement, that April 20 was a winter night and that most asthmatics believe cold triggers attacks, is not borne out by the temperatures which come from the Weather Bureau. I do agree that the second type of inversion mentioned later in this letter is always accompanied by a cold (usually westerly) change, and the mechanism as I see it is described. The temperature on April 20 in Brisbane showed a maximum of 75·4° with a

[1] *Ann. Allergy*, 1950, 8: 641.

[2] *Publ. Hlth. Rep.* (*Wash.*), 1960, 75: 307 (April).

minimum of 59·6° F. The average maximum temperature in Brisbane for the whole month of April was 78·8° F., the average minimum for the month 61·4° F., so that the temperature of April 20 was about average for the latter part of the month. Further, the average winter temperature in Brisbane is 60·6° F. maximum and 48·5° F. minimum, so that April 20 was an average day for the time of the year.

With regard to the last paragraph of Dr. Rogers' letter, I never suggested that the smog theory was new, nor did I advance any other theory at that time but that a temperature inversion triggered off spasmodic asthma.

In the twelve months' period since the original article was written, I have been able to prove to my own satisfaction that asthmatics are triggered off by temperature inversions. There has been a series of temperature inversions, lasting an average of three days, recorded in Brisbane since March this year, and the first major one was on April 22. That night again hundreds of asthmatics triggered off in the Brisbane area. I have used a small series of controls, and I will publish the results as a separate paper. It is quite possible for asthmatics to stop themselves triggering off by watching the weather maps in the capital cities on television, and in the country by watching the main city dailies, making allowance for the fact that the television weather story is up to date.

When a cold front comes through from Western Australia (which takes about seven days to cross Australia), I find that by giving the patients ephedrine, or an ephedrine-like substance, 12 hours before the inversion reaches Brisbane, and continuing it for three days, the patients do not trigger into an attack of asthma. I have used a series of controls with these experiments; but when the publicity associated with my last article came out, the controls were not happy, and are now stopping themselves triggering by watching the weather maps either in the papers or on television.

The mechanism of the triggering appears to be that the asthmatic's nose first blocks completely with an allergic rhinitis. This causes a certain proportion of patients to have a bronchospasm; but when the cold change comes through and they breathe the cold air directly on to their lungs through their mouths, without pre-warming through their nasal passages, they trigger at that stage. I feel that this could be best investigated with asthmatics at a large public hospital, with the cooperation of the Weather Bureau in announcing when an inversion is going to occur, because there are three kinds of inversions. These are as follows:

1. As on April 20, 1959, when the inversion started at sunset and its effects were progressive, reaching a maximum in the early hours of the following morning about sunrise.

2. The type in which a stream of cool air underruns the warm air, and an inversion occurs at the meeting of these two air masses of different temperatures. (This type can be picked up by watching the cold fronts on television and the papers.)

3. A collapsing high pressure system in which the air mass from aloft descends rapidly, and as it does so increases in pressure and warms up (as happens in pumping air with a bicycle pump), and this warm air descending forms an inversion with the cool air below, but disperses on reaching the surface of the earth (Donora, 1948).

In all types, the closer the inversion is to the ground the worse the effect on the sufferer. The three types can be successfully determined by the Weather Bureau. The real test came recently when two inversions were caused by collapsing high pressure systems and a number of patients triggered with this type of inversion, and not being prepared for it were, to say the least, shattered, as no front was coming through at the time—and they had no way of knowing that they should be on their ephedrine.

All asthmatics do not trigger from this cause, but most do. Further, if they trigger with one inversion and another follows in a week's time, they do not necessarily trigger on that one, but can do so on the following inversion or even on the third—there appears to be a latent period. Chronic asthmatics with emphysema are not successful as controls, as they have permanent bronchospasm and they do get worse with inversions, but are not free between them.

An omission from my acknowledgements in my original paper was to various individual members of the Australian College of General Practitioners, who have kept me advised of the sleepless nights they spend giving adrenaline to the sufferers of this disease. These are checked against the radiosonde charts supplied to me daily by the Weather Bureau.

My reason for continuing with this correspondence is a quotation in *The Practitioner* of March, 1958, in an article by H. G. Oliver[3]: "If you have anything new to say about asthma for heaven's sake say it."

Yours, etc.,

Prudential Building,
Brisbane.
August 8, 1960.

ION MORRISON.

SPECIALISTS AND HOSPITALS.

SIR: The profession will apparently soon have to decide its attitude to full-time hospital appointments, which will probably be established on a wider scale in the next few years. It is most important to avoid a split in the unity of the profession which will lay the way open to a Government policy of divide and rule, which successfully controls the profession in England.

Perhaps the best policy is to revive and insist on the sound ethical principle that a specialist is specifically consulted by a general practitioner, who remains at least nominally in charge of a case. I usually decline to treat patients who have referred themselves to specialists (or other general practitioners), and seldom refer to specialists who will see patients without a referring letter. This applies equally to hospital and private practice. The result is more satisfactory for all concerned, including the patient, and avoids the diagnostic nightmare presented by the patient who wanders about collecting varied opinions and investigations from a number of different sources. Such patients may know no better, but the doctors who see them should.

A general practitioner has, I hope, more idea of when and where to refer a patient than the patient's friend or neighbour. A clash is seldom necessary on the subject. In particular, hospital specialists should not treat patients without a referring letter (which should receive a reply), unless they can provide a full service—that is, visit the patient at home at any time if necessary. Good relations can easily be maintained on these lines.

We are supposed to know all this already, but I write because many apparently do not. We should all avoid being anybody's "other doctor".

Yours, etc.,

1029 Pittwater Road,
Collaroy, N.S.W.
August 10, 1960.

D. GARRETT.

ORGANIZATION OF THE PROFESSION.

SIR: The outcry over "crown scripts", the dearth of facilities for specialist and refresher courses in general practice, the talk of universal surgical audit before routine laboratory tests are generally available, all underline the lack of unity and enthusiasm in our profession, and the poverty of democratic process in the country's Medical Association. Let us back Dr. Jaede and Dr. Ingle in your August 6 issue. I suggest:

1. Local associations should group along the lines of proposed new States (perhaps New England, Riverina, North Queensland, Central Queensland and the Territories would emerge as new groups). These should allow more frequent fruitful contacts than State convocations. Metropolitan subgroups could be more specialized.

2. Regional offices should be maintained with branch library and other services, with officers appointed for terms of two to four years.

3. Local, specialist, women's and other groups should be encouraged to appoint delegates to regional meetings such as the biennial North Queensland Medical Congress, which should include administrative-political discussions at least before or after the academic-social programme.

4. Local funds should be subsidized from federal level for consultation and conference between groups by visits or tours, telephone hookups, purchase of standard tape recorders and exchange of tapes.

5. Any local association should have the right to have its proposals circulated by its State or regional committee and put to the vote at least once in each two months, and each regional office the same right within the Commonwealth.

6. Such plebiscites should bind the officers concerned, subject to their right to call a general meeting on the

[3] *Practitioner*, 1958, 180: 353 (March).

subject, with provision for proxy voting and central subsidy for travelling expenses.

7. Nominations to regional and State offices should be made and seconded by local or group associations, and to federal offices by regional organizations, but voting for all offices should be general, and failing a 50% vote in favour of any important office or group, nominations should be called again and a re-vote taken. Voting should be voluntary, but a levy exacted from those not voting, to pay for the cost of any re-vote. The validity and secrecy of the poll should be guarded by methods such as those successfully adopted by the Sydney University Students' Council to break up the "rigging" attempts of 1944.

8. On matters affecting one type of practice more than another, a sectional vote should be taken. Thus, for instance, the attitude of general practitioners to the "crown dose list" would appear.

Apropos of the last point: Australia's medical and hospital subsidy scheme is widely envied. What a shame our leaders in the profession who pioneered the scheme did not think to include a similar scheme for today's dearer drugs, instead of adopting the "life-saving and disease-preventing" formula now so misused by bureaucracy for short-range "economy"! The vision of Sir Earle Page, saving in hospital-days and work-days the cost of dearer drugs, has been mummified under the paperweights of the Treasury. If the profession is irresponsible in prescribing, let there be a Government and professional drive to prescribe the cheapest, using at least as elaborate propaganda as one drug firm's advertising department, and the Commonwealth will find it can scrap all books, advisory committees to re-edit books, and the 24 clerks to check that we put the right endorsements and maxima for repeats in Queensland (goodness knows how many clerks in other States).

Yours, etc.,

D. EVERINGHAM.

P.O. Box 328,
Rockhampton.
Queensland.
August 10, 1960.

THE DETECTION OF ADVANCED CANCER.

SIR: May I submit a letter on cancer detection to do with advanced cancer in contradistinction to early cancer?

If it were possible to find by a laboratory test that a patient were suffering from inoperable cancer, unnecessary operations of exploration often soon followed by death of the patient might be avoided. Perhaps some day such a test could be used with chemical treatment of cancer to follow the effect of treatment.

Since there are many patients with advanced cancer, it might be possible to find a test indicating it by correlating the laboratory findings with those at operation or *post mortem*.

The discovery of a test for late cancer would be rewarding in its theoretical interest and value in clinical practice, and the fact that gross lesions are being studied rather than early lesions suggests that success would be possible.

Yours, etc.,

ALLAN GRANT.

4 Mawson Avenue,
East Maitland, N.S.W.
August 12, 1960.

Naval, Military and Air Force.

HONOURS.

THE award of the Australian Efficiency Decoration to 4/31903 Lieutenant-Colonel Colin Marshall Gurner, Royal Australian Army Medical Corps (Medical), is notified in the *Commonwealth of Australia Gazette*, No. 50, of July 7, 1960.

APPOINTMENTS.

THE following appointments, changes, etc., are published in the *Commonwealth of Australia Gazette*, No. 50, of July 7, 1960.

NAVAL FORCES OF THE COMMONWEALTH.

Citizen Naval Forces of the Commonwealth.

Royal Australian Naval Reserve.

To be Surgeon Captain.—Surgeon Commander Harry Douglas Raffan.

To be Surgeon Commander.—Surgeon Lieutenant Commanders John Heaton Simpson and Aretas William Overton Young.

AUSTRALIAN MILITARY FORCES.

Australian Regular Army.

Royal Australian Army Medical Corps (Medical).

3/12031 Captain J. R. Thomson is retired on the completion of his Short Service Commission, 6th May, 1960.

Citizen Military Forces.

Northern Command.

Royal Australian Army Medical Corps (Medical).—The provisional rank of 1/39225 Captain L. S. Parker is confirmed. 1/61807 Captain (provisionally) R. C. S. Jackson relinquishes the provisional rank of Captain, 4th May, 1960, is transferred to the Reserve of Officers (Royal Australian Army Medical Corps (Medical)) (Northern Command) in the honorary rank of Captain, 5th May, 1960. *To be Captain (provisionally), 22nd April, 1960*—1/61916 Donald Hugh McClymont and 1/39262 Desmond David McGuckin.

Eastern Command.

Royal Australian Army Medical Corps (Medical).—2/79316 Captain F. H. Lang is seconded whilst in the United Kingdom, 12th March, 1958. The notification respecting the transfer to the Reserve of Officers of 2/79316 Captain F. H. Lang which appeared in Executive Minute No. 30 of 1958 promulgated in Commonwealth Gazette No. 26 of 1958 is withdrawn. 2/206962 Captain (provisionally) R. D. Macdonald relinquishes the provisional rank of Captain, 7th April, 1960 and is transferred to the Reserve of Officers (Royal Australian Army Medical Corps (Medical)) (Eastern Command) and is granted the honorary rank of Captain, 8th April, 1960.

Southern Command.

Royal Australian Army Medical Corps (Medical).—3/129438 Captain B. D. Appleby is seconded whilst in the United States of America, 6th February, 1960.

Central Command.

Royal Australian Army Medical Corps (Medical).—The following officers are seconded whilst in the United Kingdom:—4/32054 Captain L. C. Hoff, 21st January, 1959, and 4/32074 Captain (provisionally) A. J. Day, 1st May, 1960. 4/31918 Captain C. S. Kneebone is transferred to the Reserve of Officers (Royal Australian Army Medical Corps (Medical)) (Central Command), 14th April, 1960. The provisional appointment of 4/32074 Captain A. J. Day is terminated 30th April, 1960. *To be Captain (provisionally), 1st May, 1960*—4/32074 Allan John Day.

Western Command.

Royal Australian Army Medical Corps (Medical).—5/26582 Captain (provisionally) D. N. Spence relinquishes the provisional rank of Captain, 13th March, 1960, and is transferred to the Reserve of Officers (Royal Australian Army Medical Corps (Medical)) (Western Command) and is granted the honorary rank of Captain, 14th March, 1960. 5/26560 Captain (provisionally) D. Golinger relinquishes the provisional rank of Captain, 4th April, 1960, and is transferred to the Reserve of Officers (Royal Australian Army Medical Corps (Medical)) (Western Command) and is granted the honorary rank of Captain, 5th April, 1960.

Tasmania Command.

Royal Australian Army Medical Corps (Medical).—6/15421 Captain (provisionally) K. S. Goulston relinquishes the provisional rank of Captain, 27th April, 1960, and is transferred to the Reserve of Officers (Royal Australian Army Medical Corps (Medical)) (Tasmania Command) in the honorary rank of Captain, 28th April, 1960.

The following appointments, changes, etc., are published in the *Commonwealth of Australia Gazette*, No. 51, of July 14, 1960.

NAVAL FORCES OF THE COMMONWEALTH.

Permanent Naval Forces of the Commonwealth (Sea-Going Forces).

Appointments.—Surgeon Commander John Howard Mitchell is appointed on loan from the Royal Navy, with seniority in rank of 30th June, 1952, dated 14th April, 1960.

Fixing Rates of Pay.—Surgeon Lieutenant Victor St. Clair Dudgeon Logan is to be paid the rates of pay and allowances prescribed for Surgeon Lieutenant-Commander, whilst acting in that rank, dated 17th February, 1960.

Termination of Appointment.—The appointment of Joseph Raymond Clark as Surgeon Lieutenant (for Short Service) is terminated, dated 18th April, 1960.

Citizen Naval Forces of the Commonwealth.

Royal Australian Naval Reserve.

Promotions.—Surgeon Lieutenants John Barry Sturrock, Robert Noel Tinning and Robert Binks are promoted to the rank of Surgeon Lieutenant-Commander, dated 19th February, 1960, 5th April, 1960, and 7th April, 1960, respectively.

Resignations.—The resignation of Richard Howell Stanistreet of his appointment as Surgeon Lieutenant is accepted, dated 1st April, 1960.

ROYAL AUSTRALIAN AIR FORCE.

Permanent Air Force.

Medical Branch.

Braham Antony Pearlman (0219953) is appointed to a short-service commission, on probation for a period of twelve months, 29th February, 1960, with the rank of Flight Lieutenant.

Flight Lieutenant W. H. Taylor (035272) is granted the acting rank of Squadron Leader, 6th May, 1960.

Air Force Reserve.

Medical Branch.

The following Air Cadets are provisionally appointed to a commission, 1st July, 1960, with the rank of Pilot Officer:—Jeffrey Adrian Feint (0314537), Otto Peter Gutauskas (0314518), John Duncan Mathews (0314519), Roy Francis Richards (0314306).

Flight Lieutenant P. L. T. Ilbery (422957) is promoted to the temporary rank of Squadron Leader, 9th May, 1960.

Notes and News.

The New South Wales College of Nursing.

The impact of the need for post-graduate education in nursing is being felt throughout the nursing world, with student numbers increasing each year. The New South Wales College of Nursing is making an effort to raise a building fund of £30,000, in order that a new College may be established which will provide all the teaching facilities required. To help augment this Building Fund, St. Anne's Hospital, Marion Street, Killara, is holding a barbecue-dance, commencing at 6 p.m., on Friday, September 2, 1960. All doctors, their families and friends are invited to attend this function.

Recordati International Prize for Cardiology.

"Recordati" Laboratories have offered an international prize for cardiology to the value of 2000 dollars (U.S.), which will be awarded at the fourth World Congress in Cardiology (Mexico City, 1962) for an unpublished work on a cardiological subject. Works dealing with diagnostics, prophylaxis and therapy will be especially welcome; but consideration will be given to any work bearing on cardiology in its widest sense. Selection will be made by an international jury. The prize is open to members of the medical profession of any nationality under the age of 40 years. In the event of a tie, preference will be given to youth. Six copies of the manuscript, typed in double spacing, of not more than 50 pages in length, written in French and English and carrying not more than 10 figures suitable for the preparation of blocks of not more than 70 sq. cm., must reach the Recordati International Prize Secretariat, Via Civitali 1, Milan, Italy, not later than June 30, 1961. Entries are to be sent by

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED JULY 23, 1960.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.	Australian Capital Territory.	Australia.
Acute Rheumatism	1	..	1	..	..	..	..	..	2
Amœbiasis	..	..	..	..	..	..	..	..	..
Ancylostomiasis	..	..	..	..	..	..	3	..	3
Anthrax	..	..	..	..	..	..	..	..	..
Bilharziasis	..	..	..	..	..	..	..	..	..
Brucellosis	1	..	..	..	..	..	..	..	1
Cholera	..	..	..	..	..	..	..	..	..
Chorea (St. Vitus)	..	..	..	..	..	..	..	..	..
Dengue	..	..	..	..	..	..	..	..	..
Diarrhœa (Infantile)	14(4)	11(10)	2(2)	..	..	1	8	1	37
Diphtheria	1	..	..	..	..	..	..	..	1
Dysentery (Bacillary)	..	1(1)	3(1)	11	..	..	..	..	15
Encephalitis	..	..	..	1(1)	..	..	..	..	1
Filariasis	..	..	..	..	..	..	..	..	..
Homologous Serum Jaundice	..	..	..	..	..	..	..	..	..
Hydatid	..	1	..	..	..	..	..	..	1
Infective Hepatitis	72(30)	50(10)	8(3)	18(9)	5(1)	1(1)	1	..	155
Lead Poisoning	..	..	..	..	..	..	..	..	..
Leprosy	..	..	..	..	..	..	1	..	1
Leptospirosis	..	..	1	..	..	..	..	..	1
Malaria	..	..	..	..	1(1)	..	..	..	1
Meningococcal Infection	2	3(2)	..	..	..	..	..	..	5
Ophthalmia	..	..	..	..	..	..	..	..	..
Ornithosis	..	..	..	..	..	..	..	..	..
Paratyphoid	..	..	..	..	..	..	..	..	..
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	..	..	..	..	1	..	..	..	1
Puerperal Fever	2(1)	..	..	..	..	..	..	..	2
Rubella	..	4(1)	..	2	2(1)	..	..	..	8
Salmonella Infection	..	..	..	..	..	..	..	..	..
Scarlet Fever	8(5)	6(6)	2(2)	2(2)	1	3(1)	1	..	23
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	..	..	1	..	1(1)	..	..	..	2
Trachoma	..	..	..	..	1(1)	..	8	..	9
Trichinosis	..	..	..	..	..	..	..	..	..
Tuberculosis	..	10(7)	12(6)	12(10)	7(4)	3(1)	..	1	45
Typhoid Fever	..	..	..	..	..	..	1	..	1
Typhus (Flea-, Mite- and Tick-borne)	..	..	1	..	..	..	..	..	1
Typhus (Louse-borne)	..	..	..	..	..	..	..	..	..
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] Figures in parentheses are those for the metropolitan area.

registered mail and enclosed in two envelopes. The outer envelope will be destroyed on arrival. The inner envelope, sealed, must bear no sign of identity. It will contain (*a*) six copies of the manuscript, each bearing the same motto, (*b*) a sealed envelope bearing no marks except the above motto. This envelope is to contain name, address, motto and *curriculum vitæ* of the author, together with a declaration signed by him to the effect that the work has not been published. Further information may be obtained from the Prize Secretariat.

Johnson and Johnson Pty. Ltd.

Mr. Clarence Edwards, Chairman of Directors, has resigned from the Board of Johnson and Johnson Pty. Ltd., for reasons of health. Mr. Edwards has been a Director since the incorporation of the Australian company in 1931, and has been Chairman since 1948. Mr. W. H. Northam, who has been Managing Director since 1945, has succeeded Mr. Edwards as Chairman of the Board. Mr. R. B. Crawford, previously General Manager, has now been appointed Managing Director.

Notice.

NEW SOUTH WALES ASSOCIATION FOR MENTAL HEALTH AND NATIONAL GUIDANCE COUNCIL OF AUSTRALIA.

THE New South Wales Association for Mental Health and the National Marriage Guidance Council of Australia invite all medical practitioners to a meeting of people from the professions of medicine, law, the church, education and social work, to be held on September 1, 1960, at 8 p.m. at I.C.I. House, East Circular Quay. Dr. D. C. Maddison, Senior Lecturer in Psychiatry, University of Sydney, will speak on "Patterns of Illness in the Family", and Dr. W. L. Carrington, President, Marriage Guidance Council of Victoria, will speak on "Patterns of Therapy for the Family". These talks will be followed by a discussion with Mr. D. M. Selby, Challis Lecturer in Divorce, University of Sydney, in the chair.

Nominations and Elections.

THE following have applied for election as members of the New South Wales Branch of the British Medical Association:

Coy, Lawrence Bernard, M.B., B.S., 1958 (Univ. Sydney), 214 Barker Street, Randwick.

Hagan, Brian Elwin, M.B., B.S., 1951 (Univ. Sydney), 1/81 Birriga Road, Bellevue Hill.

Jones, Donald George, M.B., B.S., 1959 (Univ. Sydney), Lewisham Hospital, Lewisham.

Nemeth, William, M.B., B.S., 1959 (Univ. Sydney), Lewisham Hospital, Lewisham.

Papp, Peter, M.D., 1955 (Univ. Pecs.) licensed under Section 21c (3), *Medical Practitioners Act*, 1938-1958, 108 Laurel Street, Willoughby.

Deaths.

THE following deaths have been announced:

BEATTIE.—George Riddell Beattie, on August 9, 1960, at Hobart.

DICKEY.—William Craufuird McNaghten Dickey, on August 11, 1960, at Sydney.

GREGERSON.—Gerald Gregerson, on August 13, 1960, at Fitzroy, S.A.

Diary for the Month.

SEPTEMBER 1.—South Australian Branch, B.M.A.: Council Meeting.
SEPTEMBER 1.—Queensland Branch, B.M.A.: Bancroft Oration.
SEPTEMBER 2.—Queensland Branch, B.M.A.: Annual Fork Dinner.
SEPTEMBER 3.—Queensland Branch, B.M.A.: Annual General Meeting; Jackson Lecture; Sixth Branch Convocation.
SEPTEMBER 6.—New South Wales Branch, B.M.A.: Organization and Science Committee.
SEPTEMBER 7.—Western Australian Branch, B.M.A.: Branch Council Meeting.
SEPTEMBER 8.—New South Wales Branch, B.M.A.: Public Relations Committee.
SEPTEMBER 9.—Tasmanian Branch, B.M.A.: Branch Council Meeting.
SEPTEMBER 9.—Queensland Branch, B.M.A.: Council Meeting.
SEPTEMBER 13.—New South Wales Branch, B.M.A.: Executive and Finance Committee.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney): All contract practice appointments in New South Wales.

South Australian Branch (Honorary Secretary, 80 Brougham Place, North Adelaide): All contract practice appointments in South Australia.

Editorial Notices.

ALL articles submitted for publication in this Journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations, other than those normally used by the Journal, and not to underline either words or phrases.

Authors of papers are asked to state for inclusion in the title their principal qualifications as well as their relevant appointment and/or the unit, hospital or department from which the paper comes.

References to articles and books should be carefully checked. In a reference to an article in a journal the following information should be given: surname of author, initials of author, year, full title of article, name of journal, volume, number of first page of article. In a reference to a book the following information should be given: surname of author, initials of author, year of publication, full title of book, publisher, place of publication, page number (where relevant). The abbreviations used for the titles of journals are those of the list known as "World Medical Periodicals" (published by the World Medical Association). If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors submitting illustrations are asked, if possible, to provide the originals (not photographic copies) of line drawings, graphs and diagrams, and prints from the original negatives of photomicrographs. Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary is stated.

All communications should be addressed to the Editor, THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2-3.)

Members and subscribers are requested to notify the Manager, THE MEDICAL JOURNAL OF AUSTRALIA, Seamer Street, Glebe, New South Wales, without delay, of any irregularity in the delivery of this Journal. The management cannot accept any responsibility or recognize any claim arising out of non-receipt of journals unless such notification is received within one month.

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